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dis hist

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     FILE 'APOLLIT, MEDLINE, BIOSIS, EMBASE, BABS, CAPLUS, CBNB, CIN,
     COMPENDEX, DISSABS, EMA, IFIPAT, NTIS, PASCAL, PROMT, RAPRA, SCISEARCH,
     TEXTILETECH, USPATFULL, USPATOLD, USPAT2, WPIFV, WPINDEX, WSCA,
     WTEXTILES' ENTERED AT 15:26:19 ON 02 NOV 2007
L1        192059 S NEUROLOGICAL AND DISORDER
L2          3665 S L1 AND (MANNOSE OR GULOSE OR GLUCOSE-6-PHOSPHATE)
L3          1090 S L2 AND (CAMP AND MODULATOR)
L4            11 S L3 AND ONCOMODULIN
L5          1062 S L3 AND (STROKE OR ANEURISM OR SPINAL OR PARKINSON OR SCLEROS
L6           832 S L5 AND MACROPHAGE
L7           832 S L6 AND FACTOR
L8           661 S L7 AND TGF
L9           620 S L8 AND ALZHEIMER
L10           10 S L9 AND ONCOMODULIN
L11          619 S L9 AND NEURON?
L12          408 S L8 AND GLAUCOMA
L13          368 S L12 AND INTRAOCULAR
L14          367 S L13 AND INJECT?
L15         6805 S L1 AND RETINA?
L16          358 S L14 AND RETINA?
L17            5 S L10 AND MACULAR

     FILE 'CAPLUS' ENTERED AT 15:43:13 ON 02 NOV 2007
L18           69 S BENOWITZ LARRY I?/AU
L19            5 S L18 AND NEUROLOGICAL
L20           24 S L1 AND MANNOSE
L21            2 S L1 AND GULOSE
L22           16 S L1 AND GLUCOSE-6-PHOSPHATE
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NEWS 3	JUL 02	SCISEARCH enhanced with complete author names
NEWS 4	JUL 02	CHEMCATS accession numbers revised
NEWS 5	JUL 02	CA/CAplus enhanced with utility model patents from China
NEWS 6	JUL 16	CAplus enhanced with French and German abstracts
NEWS 7	JUL 18	CA/CAplus patent coverage enhanced
NEWS 8	JUL 26	USPATFULL/USPAT2 enhanced with IPC reclassification
NEWS 9	JUL 30	USGENE now available on STN
NEWS 10	AUG 06	CAS REGISTRY enhanced with new experimental property tags
NEWS 11	AUG 06	FSTA enhanced with new thesaurus edition
NEWS 12	AUG 13	CA/CAplus enhanced with additional kind codes for granted patents
NEWS 13	AUG 20	CA/CAplus enhanced with CAS indexing in pre-1907 records
NEWS 14	AUG 27	Full-text patent databases enhanced with predefined patent family display formats from INPADOCDB
NEWS 15	AUG 27	USPATOLD now available on STN
NEWS 16	AUG 28	CAS REGISTRY enhanced with additional experimental spectral property data
NEWS 17	SEP 07	STN AnaVist, Version 2.0, now available with Derwent World Patents Index
NEWS 18	SEP 13	FORIS renamed to SOFIS
NEWS 19	SEP 13	INPADOCDB enhanced with monthly SDI frequency
NEWS 20	SEP 17	CA/CAplus enhanced with printed CA page images from 1967-1998
NEWS 21	SEP 17	CAplus coverage extended to include traditional medicine patents
NEWS 22	SEP 24	EMBASE, EMBAL, and LEMBASE reloaded with enhancements
NEWS 23	OCT 02	CA/CAplus enhanced with pre-1907 records from Chemisches Zentralblatt
NEWS 24	OCT 19	BEILSTEIN updated with new compounds

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                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     1.05       1.05

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FILE 'IFIPAT' ENTERED AT 15:26:19 ON 02 NOV 2007
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FILE 'PROMT' ENTERED AT 15:26:19 ON 02 NOV 2007

FILE 'RAPRA' ENTERED AT 15:26:19 ON 02 NOV 2007

FILE 'SCISEARCH' ENTERED AT 15:26:19 ON 02 NOV 2007

FILE 'TEXTILETECH' ENTERED AT 15:26:19 ON 02 NOV 2007

FILE 'USPATFULL' ENTERED AT 15:26:19 ON 02 NOV 2007

FILE 'USPATOLD' ENTERED AT 15:26:19 ON 02 NOV 2007

FILE 'USPAT2' ENTERED AT 15:26:19 ON 02 NOV 2007

FILE 'WPIDS' ACCESS NOT AUTHORIZED

FILE 'WPIFV' ENTERED AT 15:26:19 ON 02 NOV 2007

FILE 'WPINDEX' ENTERED AT 15:26:19 ON 02 NOV 2007

FILE 'WSCA' ENTERED AT 15:26:19 ON 02 NOV 2007

FILE 'WTEXTILES' ENTERED AT 15:26:19 ON 02 NOV 2007


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=> s neurological and disorder
L1      192059 NEUROLOGICAL AND DISORDER

=> s l1 and (mannose or gulose or glucose-6-phosphate)
  17 FILES SEARCHED...
L2        3665 L1 AND (MANNOSE OR GULOSE OR GLUCOSE-6-PHOSPHATE)

=> s l2 and (cAMP and modulator)
L3        1090 L2 AND (CAMP AND MODULATOR)

=> s l3 and oncomodulin
L4          11 L3 AND ONCOMODULIN

=> dis l4 1-11 bi abs
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L4   ANSWER 1 OF 11  IFIPAT  COPYRIGHT 2007 IFI on STN
AN    11017314  IFIPAT;IFIUDB;IFICDB
TI    METHODS AND COMPOSITIONS FOR TREATMENT OF NEUROLOGICAL
```

DISORDER
INF Benowitz; Larry I., Newton, MA, US
IN Benowitz Larry I
PAF Children's Medical Center Corporation, Boston, MA, US
PA Children's Medical Center Corp The (10709)
AG DAVID S. RESNICK, 100 SUMMER STREET, NIXON PEABODY LLP, BOSTON, MA, 02110-2131, US
PI US 2005256059 A1 20051117
AI US 2003-528685 20030925
WO 2003-US30466 20030925
20050718 PCT 371 date
20050718 PCT 102(e) date
PRAI US 2002-414063P 20020927 (Provisional)
FI US 2005256059 20051117
DT Utility; Patent Application - First Publication
FS CHEMICAL
APPLICATION
ED Entered STN: 18 Nov 2005
Last Updated on STN: 18 Nov 2005
CLMN 26

L4 ANSWER 2 OF 11 USPATFULL on STN
AN 2006:215041 USPATFULL
TI Polynucleotide encoding a novel cysteine protease of the calpain superfamily, CAN-12, and variants thereof
IN Chen, Jian, Princeton, NJ, UNITED STATES
Feder, John N., Belle Mead, NJ, UNITED STATES
Nelson, Thomas C., Lawrenceville, NJ, UNITED STATES
Seiler, Steven, Pennington, NJ, UNITED STATES
Vaz, Roy J., North Branch, NJ, UNITED STATES
Duclos, Franck, Washington Crossing, PA, UNITED STATES
PI US 2006183196 A1 20060817
AI US 2006-407134 A1 20060419 (11)
RLI Division of Ser. No. US 2002-116519, filed on 3 Apr 2002, PENDING
PRAI US 2001-281253P 20010403 (60)
US 2001-288768P 20010504 (60)
US 2001-296180P 20010606 (60)
US 2001-300620P 20010625 (60)
DT Utility
FS APPLICATION
LREP LOUIS J. WILLE, BRISTOL-MYERS SQUIBB COMPANY, PATENT DEPARTMENT, P O BOX 4000, PRINCETON, NJ, 08543-4000, US
CLMN Number of Claims: 24
ECL Exemplary Claim: 1-23
DRWN 27 Drawing Page(s)
LN.CNT 29767
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 3 OF 11 USPATFULL on STN
AN 2005:293510 USPATFULL
TI Methods and compositions for treatment of neurological disorder
IN Benowitz, Larry I., Newton, MA, UNITED STATES
PA Children's Medical Center Corporation, Boston, MA, UNITED STATES (U.S. corporation)
PI US 2005256059 A1 20051117
AI US 2003-528685 A1 20030925 (10)
WO 2003-US30466 20030925
20050718 PCT 371 date
PRAI US 2002-414063P 20020927 (60)
DT Utility
FS APPLICATION
LREP DAVID S. RESNICK, 100 SUMMER STREET, NIXON PEABODY LLP, BOSTON, MA, 02110-2131, US

CLMN Number of Claims: 26
ECL Exemplary Claim: 1
DRWN 14 Drawing Page(s)
LN.CNT 1625
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 4 OF 11 USPATFULL on STN
AN 2005:69453 USPATFULL
TI Methods and compositions for producing a neurosalutary effect in a subject
IN Benowitz, Larry I., Newton Square, MA, UNITED STATES
PI US 2005059594 A1 20050317
AI US 2004-894351 A1 20040719 (10)
RLI Continuation of Ser. No. US 2001-872347, filed on 1 Jun 2001, ABANDONED
PRAI US 2000-208778P 20000601 (60)
DT Utility
FS APPLICATION
LREP David S. Resnick, NIXON PEABODY LLP, 100 Summer Street, Boston, MA, 02110
CLMN Number of Claims: 46
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1373
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 5 OF 11 USPATFULL on STN
AN 2004:44514 USPATFULL
TI Polynucleotides encoding novel human mitochondrial and microsomal glycerol-3-phosphate acyl-transferases and variants thereof
IN Farrelly, Dennis, Monmouth Junction, NJ, UNITED STATES
Chen, Jian, Princeton, NJ, UNITED STATES
Nelson, Thomas C., Lawrenceville, NJ, UNITED STATES
Feder, John N., Belle Mead, NJ, UNITED STATES
Wu, Shujian, Langhorne, PA, UNITED STATES
Bassolino, Donna A., Hamilton, NJ, UNITED STATES
Krystek, Stanley R., Ringoes, NJ, UNITED STATES
PI US 2004033506 A1 20040219
AI US 2002-308128 A1 20021202 (10)
PRAI US 2001-334904P 20011130 (60)
DT Utility
FS APPLICATION
LREP STEPHEN B. DAVIS, BRISTOL-MYERS SQUIBB COMPANY, PATENT DEPARTMENT, P O BOX 4000, PRINCETON, NJ, 08543-4000
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN 37 Drawing Page(s)
LN.CNT 28557
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 6 OF 11 USPATFULL on STN
AN 2004:18791 USPATFULL
TI Polynucleotide encoding a novel cysteine protease of the calpain superfamily, Protease-42
IN Duclos, Franck, Washington Crossing, PA, UNITED STATES
Chen, Jian, Princeton, NJ, UNITED STATES
Feder, John N., Belle Mead, NJ, UNITED STATES
Nayeem, Akbar, Newtown, PA, UNITED STATES
Nelson, Thomas C., Lawrenceville, NJ, UNITED STATES
PI US 2004014093 A1 20040122
AI US 2003-390585 A1 20030314 (10)
PRAI US 2002-364941P 20020314 (60)
DT Utility
FS APPLICATION
LREP STEPHEN B. DAVIS, BRISTOL-MYERS SQUIBB COMPANY, PATENT DEPARTMENT, P O

BOX 4000, PRINCETON, NJ, 08543-4000
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN 19 Drawing Page(s)
LN.CNT 19269
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 7 OF 11 USPATFULL on STN
AN 2003:166515 USPATFULL
TI Polynucleotide encoding a novel cysteine protease of the calpain superfamily, CAN-12, and variants thereof
IN Chen, Jian, Princeton, NJ, UNITED STATES
Feder, John N., Belle Mead, NJ, UNITED STATES
Nelson, Thomas C., Lawrenceville, NJ, UNITED STATES
Seiler, Steven, Pennington, NJ, UNITED STATES
Vaz, Roy J., North Branch, NJ, UNITED STATES
Duclos, Franck, Washington Crossing, PA, UNITED STATES
PI US 2003114373 A1 20030619
US 7186564 B2 20070306
AI US 2002-116519 A1 20020403 (10)
PRAI US 2001-281253P 20010403 (60)
US 2001-288768P 20010504 (60)
US 2001-296180P 20010606 (60)
US 2001-300620P 20010625 (60)
DT Utility
FS APPLICATION
LREP STEPHEN B. DAVIS, BRISTOL-MYERS SQUIBB COMPANY, PATENT DEPARTMENT, P O BOX 4000, PRINCETON, NJ, 08543-4000
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN 27 Drawing Page(s)
LN.CNT 30149
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 8 OF 11 USPATFULL on STN
AN 2002:221781 USPATFULL
TI Methods and compositions for producing a neurosalutary effect in a subject
IN Benowitz, Larry I., Newton Square, MA, UNITED STATES
PI US 2002119923 A1 20020829
AI US 2001-872347 A1 20010601 (9)
PRAI US 2000-208778P 20000601 (60)
DT Utility
FS APPLICATION
LREP LAHIVE & COCKFIELD, 28 STATE STREET, BOSTON, MA, 02109
CLMN Number of Claims: 46
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1372
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 9 OF 11 USPAT2 on STN
AN 2003:166515 USPAT2
TI Polynucleotides encoding novel cysteine proteases of the calpain superfamily, CAN-12v1 and CAN-12v2.
IN Chen, Jian, Princeton, NJ, UNITED STATES
Nelson, Thomas C., Lawrenceville, NJ, UNITED STATES
Vaz, Roy J., North Branch, NJ, UNITED STATES
Duclos, Franck, Washington Crossing, PA, UNITED STATES
PA Bristol-Myers Squibb Company, Princeton, NJ, UNITED STATES (U.S. corporation)
PI US 7186564 B2 20070306
AI US 2002-116519 20020403 (10)
PRAI US 2001-300620P 20010625 (60)

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       US 2001-296180P     20010606 (60)
       US 2001-288768P     20010504 (60)
       US 2001-281253P     20010403 (60)
DT     Utility
FS     GRANTED
EXNAM  Primary Examiner: Nashed, Nashaat T.; Assistant Examiner: Moore, William
       W.
LREP   D'Amico, Stephen C.
CLMN   Number of Claims: 18
ECL    Exemplary Claim: 1
DRWN   27 Drawing Figure(s); 27 Drawing Page(s)
LN.CNT 30048
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4   ANSWER 10 OF 11  WPINDEX COPYRIGHT 2007       THE THOMSON CORP on STN
AN   2005-521956 [53]   WPINDEX
DNN  N2005-426370 [53]
TI   Stimulating axonal growth of central nervous system (CNS) neurons,
     involves contacting CNS neurons with nogo receptor antagonist, and
     contacting CNS neurons with agent that activates growth pathway of CNS
     neurons
DC   S03
IN   BENOWITZ L I; FISCHER D; BENOWITZ L
PA   (CHIL-N) CHILDRENS MEDICAL CENT
CYC  107
PIA  WO 2005059515   A2 20050630 (200553)* EN  74[9]
     EP 1695061      A2 20060830 (200657)  EN
     JP 2007514748   W  20070607 (200739)  JA  49
ADT  WO 2005059515 A2 WO 2004-US42255 20041216; EP 1695061 A2 EP 2004-814439
     20041216; EP 1695061 A2 WO 2004-US42255 20041216; JP 2007514748 W WO
     2004-US42255 20041216; JP 2007514748 W JP 2006-545428 20041216
FDT  EP 1695061      A2 Based on WO 2005059515   A; JP 2007514748   W Based on
     WO 2005059515   A
PRAI US 2003-529833P 20031216

L4   ANSWER 11 OF 11  WPINDEX COPYRIGHT 2007       THE THOMSON CORP on STN
AN   2004-316013 [29]   WPINDEX
DNC  C2004-119849 [29]
TI   Use of hexose (e.g. D-mannose) to treat/alleviate
     neurological disorders such as traumatic brain injury,
     stroke, cerebral aneurysm, Parkinson's disease, amyotrophic lateral
     sclerosis and Alzheimer's disease
DC   B03; B04
IN   BENOWITZ L I
PA   (CHIL-N) CHILDRENS MEDICAL CENT
CYC  104
PIA  WO 2004028468   A2 20040408 (200429)* EN  59[9]
     AU 2003272728   A1 20040419 (200462)  EN
     EP 1542702      A2 20050622 (200541)  EN
     US 20050256059  A1 20051117 (200576)  EN
     JP 2006503847   W  20060202 (200611)  JA  35
     AU 2003272728   A8 20051103 (200629)  EN
     CN 1703227      A  20051130 (200636)  ZH
ADT  WO 2004028468 A2 WO 2003-US30466 20030925; US 20050256059 A1 Provisional
     US 2002-414063P 20020927; AU 2003272728 A1 AU 2003-272728 20030925; AU
     2003272728 A8 AU 2003-272728 20030925; EP 1542702 A2 EP 2003-754929
     20030925; EP 1542702 A2 WO 2003-US30466 20030925; US 20050256059 A1 WO
     2003-US30466 20030925; JP 2006503847 W WO 2003-US30466 20030925; JP
     2006503847 W JP 2004-540004 20030925; US 20050256059 A1 US 2005-528685
     20050718; CN 1703227 A CN 2003-825428 20030925
FDT  AU 2003272728   A1 Based on WO 2004028468   A; EP 1542702      A2 Based on
     WO 2004028468   A; JP 2006503847   W Based on WO 2004028468   A; AU
     2003272728   A8 Based on WO 2004028468   A
PRAI US 2002-414063P 20020927
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US 2005-528685 20050718

=> dis hist

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FILE 'APOLLIT, MEDLINE, BIOSIS, EMBASE, BABS, CAPLUS, CBNB, CIN, COMPENDEX, DISSABS, EMA, IFIPAT, NTIS, PASCAL, PROMT, RAPRA, SCISEARCH, TEXTILETECH, USPATFULL, USPATOLD, USPAT2, WPIFV, WPINDEX, WSCA, WTEXTILES' ENTERED AT 15:26:19 ON 02 NOV 2007

L1 192059 S NEUROLOGICAL AND DISORDER
L2 3665 S L1 AND (MANNOSE OR GULOSE OR GLUCOSE-6-PHOSPHATE)
L3 1090 S L2 AND (CAMP AND MODULATOR)
L4 11 S L3 AND ONCOMODULIN

=> s l3 and (stroke or aneurism or spinal or parkinson's or sclerosis or dementia or Picks or huntington or shy-dranger or atropy or Gilles or Meige or parapleg?)
MISMATCHED QUOTE 'PARKINSON'S'
Quotation marks (or apostrophes) must be used in pairs,
one before and one after the expression you are setting
off or masking.

=> s l3 and (stroke or aneurism or spinal or parkinson or sclerosis or dementia or Picks or huntington or shy-dranger or atropy or Gilles or Meige or parapleg?)
19 FILES SEARCHED...
L5 1062 L3 AND (STROKE OR ANEURISM OR SPINAL OR PARKINSON OR SCLEROSIS OR DEMENTIA OR PICKS OR HUNTINGTON OR SHY-DRANGER OR ATROPY OR GILLES OR MEIGE OR PARAPLEG?)

=> s l5 and macrophage
L6 832 L5 AND MACROPHAGE

=> s l6 and factor
24 FILES SEARCHED...
L7 832 L6 AND FACTOR

=> s l7 and TGF
L8 661 L7 AND TGF

=> s l8 and Alzheimer
L9 620 L8 AND ALZHEIMER

=> s l9 and oncomodulin
L10 10 L9 AND ONCOMODULIN

=> s l9 and neuron?
L11 619 L9 AND NEURON?

=> s l8 and glaucoma
L12 408 L8 AND GLAUCOMA

=> s l12 and intraocular
L13 368 L12 AND INTRAOCULAR

=> s l13 and inject?
L14 367 L13 AND INJECT?

=> s l1 and retina?
L15 6805 L1 AND RETINA?

=> s l14 and retina?
L16 358 L14 AND RETINA?

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=> s l10 and macular
L17          5 L10 AND MACULAR

=> dis l17 1-5 bib abs

L17  ANSWER 1 OF 5  IFIPAT  COPYRIGHT 2007 IFI on STN
AN    11017314  IFIPAT;IFIUDB;IFICDB
TI    METHODS AND COMPOSITIONS FOR TREATMENT OF NEUROLOGICAL
      DISORDER
INF   Benowitz; Larry I., Newton, MA, US
IN    Benowitz Larry I
PAF   Children's Medical Center Corporation, Boston, MA, US
PA    Children's Medical Center Corp The (10709)
AG    DAVID S. RESNICK, 100 SUMMER STREET, NIXON PEABODY LLP, BOSTON, MA,
      02110-2131, US
PI    US 2005256059   A1  20051117
AI    US 2003-528685      20030925
      WO 2003-US30466     20030925
                          20050718  PCT 371 date
                          20050718  PCT 102(e) date
PRAI  US 2002-414063P     20020927 (Provisional)
FI    US 2005256059       20051117
DT    Utility; Patent Application - First Publication
FS    CHEMICAL
      APPLICATION
ED    Entered STN: 18 Nov 2005
      Last Updated on STN: 18 Nov 2005
CLMN  26
AB    The present invention provides methods and compositions for producing a
      neurosalutary effect in a subject useful in treatment of
      neurological disorders, including retinal and optic
      nerve damage, in a subject in need thereof. The method includes
      administering to a subject a therapeutically effective amount of a
      hexose, such as mannose.
CLMN  26

L17  ANSWER 2 OF 5  USPATFULL on STN
AN     2005:293510  USPATFULL
TI     Methods and compositions for treatment of neurological
       disorder
IN     Benowitz, Larry I., Newton, MA, UNITED STATES
PA     Children's Medical Center Corporation, Boston, MA, UNITED STATES (U.S.
       corporation)
PI     US 2005256059       A1  20051117
AI     US 2003-528685      A1  20030925 (10)
       WO 2003-US30466         20030925
                               20050718  PCT 371 date
PRAI   US 2002-414063P     20020927 (60)
DT     Utility
FS     APPLICATION
LREP   DAVID S. RESNICK, 100 SUMMER STREET, NIXON PEABODY LLP, BOSTON, MA,
       02110-2131, US
CLMN   Number of Claims: 26
ECL    Exemplary Claim: 1
DRWN   14 Drawing Page(s)
LN.CNT 1625
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB     The present invention provides methods and compositions for producing a
       neurosalutary effect in a subject useful in treatment of
       neurological disorders, including retinal and optic
       nerve damage, in a subject in need thereof. The method includes
       administering to a subject a therapeutically effective amount of a
       hexose, such as mannose.
```

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L17 ANSWER 3 OF 5 USPATFULL on STN
AN 2004:44514 USPATFULL
TI Polynucleotides encoding novel human mitochondrial and microsomal glycerol-3-phosphate acyl-transferases and variants thereof
IN Farrelly, Dennis, Monmouth Junction, NJ, UNITED STATES
Chen, Jian, Princeton, NJ, UNITED STATES
Nelson, Thomas C., Lawrenceville, NJ, UNITED STATES
Feder, John N., Belle Mead, NJ, UNITED STATES
Wu, Shujian, Langhorne, PA, UNITED STATES
Bassolino, Donna A., Hamilton, NJ, UNITED STATES
Krystek, Stanley R., Ringoes, NJ, UNITED STATES
PI US 2004033506 A1 20040219
AI US 2002-308128 A1 20021202 (10)
PRAI US 2001-334904P 20011130 (60)
DT Utility
FS APPLICATION
LREP STEPHEN B. DAVIS, BRISTOL-MYERS SQUIBB COMPANY, PATENT DEPARTMENT, P O BOX 4000, PRINCETON, NJ, 08543-4000
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN 37 Drawing Page(s)
LN.CNT 28557
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides novel polynucleotides encoding Mitochondrial GPAT, Microsomal GPAT_hlog1, Microsomal GPAT_hlog2, Microsomal GPAT_hlog3, and/or Microsomal GPAT_hlog3_v1 polypeptides, fragments and homologues thereof. Also provided are vectors, host cells, antibodies, and recombinant and synthetic methods for producing said polypeptides. The invention further relates to diagnostic and therapeutic methods for applying these novel Mitochondrial GPAT, Microsomal GPAT_hlog1, Microsomal GPAT_hlog2, Microsomal GPAT_hlog3, and/or Microsomal GPAT_hlog3_v1 polypeptides to the diagnosis, treatment, and/or prevention of various diseases and/or disorders related to these polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of the polynucleotides and polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L17 ANSWER 4 OF 5 USPATFULL on STN
AN 2004:18791 USPATFULL
TI Polynucleotide encoding a novel cysteine protease of the calpain superfamily, Protease-42
IN Duclos, Franck, Washington Crossing, PA, UNITED STATES
Chen, Jian, Princeton, NJ, UNITED STATES
Feder, John N., Belle Mead, NJ, UNITED STATES
Nayeem, Akbar, Newtown, PA, UNITED STATES
Nelson, Thomas C., Lawrenceville, NJ, UNITED STATES
PI US 2004014093 A1 20040122
AI US 2003-390585 A1 20030314 (10)
PRAI US 2002-364941P 20020314 (60)
DT Utility
FS APPLICATION
LREP STEPHEN B. DAVIS, BRISTOL-MYERS SQUIBB COMPANY, PATENT DEPARTMENT, P O BOX 4000, PRINCETON, NJ, 08543-4000
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN 19 Drawing Page(s)
LN.CNT 19269
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides novel polynucleotides encoding Protease-42 polypeptides, fragments and homologues thereof. Also

provided are vectors, host cells, antibodies, and recombinant and synthetic methods for producing said polypeptides. The invention further relates to diagnostic and therapeutic methods for applying these novel Protease-42 polypeptides to the diagnosis, treatment, and/or prevention of various diseases and/or disorders related to these polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of the polynucleotides and polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L17 ANSWER 5 OF 5 WPINDEX COPYRIGHT 2007 THE THOMSON CORP on STN
AN 2004-316013 [29] WPINDEX
DNC C2004-119849 [29]
TI Use of hexose (e.g. D-mannose) to treat/alleviate neurological disorders such as traumatic brain injury, stroke, cerebral aneurysm, Parkinson's disease, amyotrophic lateral sclerosis and Alzheimer's disease
DC B03; B04
IN BENOWITZ L I
PA (CHIL-N) CHILDRENS MEDICAL CENT
CYC 104
PIA WO 2004028468 A2 20040408 (200429)* EN 59[9]
AU 2003272728 A1 20040419 (200462) EN
EP 1542702 A2 20050622 (200541) EN
US 20050256059 A1 20051117 (200576) EN
JP 2006503847 W 20060202 (200611) JA 35
AU 2003272728 A8 20051103 (200629) EN
CN 1703227 A 20051130 (200636) ZH
ADT WO 2004028468 A2 WO 2003-US30466 20030925; US 20050256059 A1 Provisional US 2002-414063P 20020927; AU 2003272728 A1 AU 2003-272728 20030925; AU 2003272728 A8 AU 2003-272728 20030925; EP 1542702 A2 EP 2003-754929 20030925; EP 1542702 A2 WO 2003-US30466 20030925; US 20050256059 A1 WO 2003-US30466 20030925; JP 2006503847 W WO 2003-US30466 20030925; JP 2006503847 W JP 2004-540004 20030925; US 20050256059 A1 US 2005-528685 20050718; CN 1703227 A CN 2003-825428 20030925
FDT AU 2003272728 A1 Based on WO 2004028468 A; EP 1542702 A2 Based on WO 2004028468 A; JP 2006503847 W Based on WO 2004028468 A; AU 2003272728 A8 Based on WO 2004028468 A
PRAI US 2002-414063P 20020927
US 2005-528685 20050718
AN 2004-316013 [29] WPINDEX
AB WO 2004028468 A2 UPAB: 20060203
NOVELTY - Treatment of a neurological disorder comprises the administration of a hexose (I).

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:

(1) an article of manufacture that comprises a pharmaceutical agent (A) (comprising D-mannose) contained within a packaging material which comprises a label indicating that (A) may be administered together with a carrier for a sufficient term at an effective dose to treat a neurological disorder; and

(2) a formulation comprising D-mannose, a cyclic adenosine monophosphate (cAMP) modulator and a carrier.

ACTIVITY - Neuroprotective; Vulnerary; Cerebroprotective; Vasotropic; Antiparkinsonian; Nootropic; CNS-Gen.; Anticonvulsant; Neuroleptic; Muscular-Gen.; Relaxant; Antiinflammatory; Ophthalmological.

The axon-promoting effects of hexose sugars and related compounds were tested on retinal ganglion cells in culture. (I) exhibited a median effective dosage value of approximately 10 microM.

MECHANISM OF ACTION - None given in the source material.

USE - Treatment with (I) reverses neuronal damage and treats/alleviates neurological disorders (preferably traumatic brain injury, stroke, cerebral aneurysm,

Parkinson's disease, amyotrophic lateral sclerosis, Alzheimer's disease, diffuse cerebral cortical atrophy, Lewy-body dementia, Pick disease, mesolimbocortical dementia, thalamic degeneration, Huntington's chorea, cortical-striatal-spinal degeneration, cortical-basal ganglionic degeneration, cerebrocerebellar degeneration, familial dementia with spastic paraparesis, polyglucosan body disease, Shy-Drager syndrome, olivopontocerebellar atrophy, progressive supranuclear palsy, dystonia musculorum deformans, Hallervorden-Spatz disease, Meige syndrome, familial tremors, Gilles de la Tourette syndrome, acanthocytic chorea, Friedreich ataxia, Holmes familial cortical cerebellar atrophy, Gerstmann-Straussler-Scheinker disease, progressive spinal muscular atrophy, progressive balbar palsy, primary lateral sclerosis, hereditary muscular atrophy, spastic paraplegia , peroneal muscular atrophy, hypertrophic interstitial polyneuropathy, heredopathia atactica polyneuritiformis, optic neuropathy, ophthalmoplegia and, particularly, spinal cord injury (characterized by monoplegia, diplegia, paraplegia, hemiplegia and quadriplegia), retinal damage (resulting from macular degeneration) or optic nerve damage (resulting from glaucoma) (all claimed).

=> dis l10 1-10 bib abs

L10 ANSWER 1 OF 10 IFIPAT COPYRIGHT 2007 IFI on STN
AN 11017314 IFIPAT;IFIUDB;IFICDB
TI METHODS AND COMPOSITIONS FOR TREATMENT OF NEUROLOGICAL DISORDER
INF Benowitz; Larry I., Newton, MA, US
IN Benowitz Larry I
PAF Children's Medical Center Corporation, Boston, MA, US
PA Children's Medical Center Corp The (10709)
AG DAVID S. RESNICK, 100 SUMMER STREET, NIXON PEABODY LLP, BOSTON, MA, 02110-2131, US
PI US 2005256059 A1 20051117
AI US 2003-528685 20030925
WO 2003-US30466 20030925
20050718 PCT 371 date
20050718 PCT 102(e) date
PRAI US 2002-414063P 20020927 (Provisional)
FI US 2005256059 20051117
DT Utility; Patent Application - First Publication
FS CHEMICAL
APPLICATION
ED Entered STN: 18 Nov 2005
Last Updated on STN: 18 Nov 2005
CLMN 26
AB The present invention provides methods and compositions for producing a neurosalutary effect in a subject useful in treatment of neurological disorders, including retinal and optic nerve damage, in a subject in need thereof. The method includes administering to a subject a therapeutically effective amount of a hexose, such as mannose.
CLMN 26

L10 ANSWER 2 OF 10 USPATFULL on STN
AN 2006:215041 USPATFULL
TI Polynucleotide encoding a novel cysteine protease of the calpain superfamily, CAN-12, and variants thereof
IN Chen, Jian, Princeton, NJ, UNITED STATES
Feder, John N., Belle Mead, NJ, UNITED STATES
Nelson, Thomas C., Lawrenceville, NJ, UNITED STATES
Seiler, Steven, Pennington, NJ, UNITED STATES
Vaz, Roy J., North Branch, NJ, UNITED STATES

Duclos, Franck, Washington Crossing, PA, UNITED STATES
PI US 2006183196 A1 20060817
AI US 2006-407134 A1 20060419 (11)
RLI Division of Ser. No. US 2002-116519, filed on 3 Apr 2002, PENDING
PRAI US 2001-281253P 20010403 (60)
US 2001-288768P 20010504 (60)
US 2001-296180P 20010606 (60)
US 2001-300620P 20010625 (60)
DT Utility
FS APPLICATION
LREP LOUIS J. WILLE, BRISTOL-MYERS SQUIBB COMPANY, PATENT DEPARTMENT, P O BOX 4000, PRINCETON, NJ, 08543-4000, US
CLMN Number of Claims: 24
ECL Exemplary Claim: 1-23
DRWN 27 Drawing Page(s)
LN.CNT 29767
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides novel polynucleotides encoding CAN-12 polypeptides, fragments and homologues thereof. The present invention also provides polynucleotides encoding variants of CAN-12 polypeptides, CAN-12v1 and CAN-12v2. Also provided are vectors, host cells, antibodies, and recombinant and synthetic methods for producing said polypeptides. The invention further relates to diagnostic and therapeutic methods for applying these novel CAN-12, CAN-12v1, and CAN-12v2 polypeptides to the diagnosis, treatment, and/or prevention of various diseases and/or disorders related to these polypeptides, particularly neuro- and musculo-degenerative conditions. The invention further relates to screening methods for identifying agonists and antagonists of the polynucleotides and polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 3 OF 10 USPATFULL on STN
AN 2005:293510 USPATFULL
TI Methods and compositions for treatment of neurological disorder
IN Benowitz, Larry I., Newton, MA, UNITED STATES
PA Children's Medical Center Corporation, Boston, MA, UNITED STATES (U.S. corporation)
PI US 2005256059 A1 20051117
AI US 2003-528685 A1 20030925 (10)
WO 2003-US30466 20030925
20050718 PCT 371 date
PRAI US 2002-414063P 20020927 (60)
DT Utility
FS APPLICATION
LREP DAVID S. RESNICK, 100 SUMMER STREET, NIXON PEABODY LLP, BOSTON, MA, 02110-2131, US
CLMN Number of Claims: 26
ECL Exemplary Claim: 1
DRWN 14 Drawing Page(s)
LN.CNT 1625
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides methods and compositions for producing a neurosalutary effect in a subject useful in treatment of neurological disorders, including retinal and optic nerve damage, in a subject in need thereof. The method includes administering to a subject a therapeutically effective amount of a hexose, such as mannose.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 4 OF 10 USPATFULL on STN

AN 2005:69453 USPATFULL
TI Methods and compositions for producing a neurosalutary effect in a subject
IN Benowitz, Larry I., Newton Square, MA, UNITED STATES
PI US 2005059594 A1 20050317
AI US 2004-894351 A1 20040719 (10)
RLI Continuation of Ser. No. US 2001-872347, filed on 1 Jun 2001, ABANDONED
PRAI US 2000-208778P 20000601 (60)
DT Utility
FS APPLICATION
LREP David S. Resnick, NIXON PEABODY LLP, 100 Summer Street, Boston, MA, 02110
CLMN Number of Claims: 46
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1373
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods and compositions for producing a neurosalutary effect in a subject, such as modulating neuronal survival and/or regeneration in a subject, are provided. Pharmaceutical and packaged formulations are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 5 OF 10 USPATFULL on STN
AN 2004:44514 USPATFULL
TI Polynucleotides encoding novel human mitochondrial and microsomal glycerol-3-phosphate acyl-transferases and variants thereof
IN Farrelly, Dennis, Monmouth Junction, NJ, UNITED STATES
Chen, Jian, Princeton, NJ, UNITED STATES
Nelson, Thomas C., Lawrenceville, NJ, UNITED STATES
Feder, John N., Belle Mead, NJ, UNITED STATES
Wu, Shujian, Langhorne, PA, UNITED STATES
Bassolino, Donna A., Hamilton, NJ, UNITED STATES
Krystek, Stanley R., Ringoes, NJ, UNITED STATES
PI US 2004033506 A1 20040219
AI US 2002-308128 A1 20021202 (10)
PRAI US 2001-334904P 20011130 (60)
DT Utility
FS APPLICATION
LREP STEPHEN B. DAVIS, BRISTOL-MYERS SQUIBB COMPANY, PATENT DEPARTMENT, P O BOX 4000, PRINCETON, NJ, 08543-4000
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN 37 Drawing Page(s)
LN.CNT 28557
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides novel polynucleotides encoding Mitochondrial GPAT, Microsomal GPAT_hlog1, Microsomal GPAT_hlog2, Microsomal GPAT_hlog3, and/or Microsomal GPAT_hlog3_v1 polypeptides, fragments and homologues thereof. Also provided are vectors, host cells, antibodies, and recombinant and synthetic methods for producing said polypeptides. The invention further relates to diagnostic and therapeutic methods for applying these novel Mitochondrial GPAT, Microsomal GPAT_hlog1, Microsomal GPAT_hlog2, Microsomal GPAT_hlog3, and/or Microsomal GPAT_hlog3_v1 polypeptides to the diagnosis, treatment, and/or prevention of various diseases and/or disorders related to these polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of the polynucleotides and polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 6 OF 10 USPATFULL on STN

AN 2004:18791 USPATFULL
TI Polynucleotide encoding a novel cysteine protease of the calpain superfamily, Protease-42
IN Duclos, Franck, Washington Crossing, PA, UNITED STATES
Chen, Jian, Princeton, NJ, UNITED STATES
Feder, John N., Belle Mead, NJ, UNITED STATES
Nayeem, Akbar, Newtown, PA, UNITED STATES
Nelson, Thomas C., Lawrenceville, NJ, UNITED STATES
PI US 2004014093 A1 20040122
AI US 2003-390585 A1 20030314 (10)
PRAI US 2002-364941P 20020314 (60)
DT Utility
FS APPLICATION
LREP STEPHEN B. DAVIS, BRISTOL-MYERS SQUIBB COMPANY, PATENT DEPARTMENT, P O BOX 4000, PRINCETON, NJ, 08543-4000
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN 19 Drawing Page(s)
LN.CNT 19269
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides novel polynucleotides encoding Protease-42 polypeptides, fragments and homologues thereof. Also provided are vectors, host cells, antibodies, and recombinant and synthetic methods for producing said polypeptides. The invention further relates to diagnostic and therapeutic methods for applying these novel Protease-42 polypeptides to the diagnosis, treatment, and/or prevention of various diseases and/or disorders related to these polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of the polynucleotides and polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 7 OF 10 USPATFULL on STN
AN 2003:166515 USPATFULL
TI Polynucleotide encoding a novel cysteine protease of the calpain superfamily, CAN-12, and variants thereof
IN Chen, Jian, Princeton, NJ, UNITED STATES
Feder, John N., Belle Mead, NJ, UNITED STATES
Nelson, Thomas C., Lawrenceville, NJ, UNITED STATES
Seiler, Steven, Pennington, NJ, UNITED STATES
Vaz, Roy J., North Branch, NJ, UNITED STATES
Duclos, Franck, Washington Crossing, PA, UNITED STATES
PI US 2003114373 A1 20030619
US 7186564 B2 20070306
AI US 2002-116519 A1 20020403 (10)
PRAI US 2001-281253P 20010403 (60)
US 2001-288768P 20010504 (60)
US 2001-296180P 20010606 (60)
US 2001-300620P 20010625 (60)
DT Utility
FS APPLICATION
LREP STEPHEN B. DAVIS, BRISTOL-MYERS SQUIBB COMPANY, PATENT DEPARTMENT, P O BOX 4000, PRINCETON, NJ, 08543-4000
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN 27 Drawing Page(s)
LN.CNT 30149
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides novel polynucleotides encoding CAN-12 polypeptides, fragments and homologues thereof. The present invention also provides polynucleotides encoding variants of CAN-12 polypeptides, CAN-12v1 and CAN-12v2. Also provided are vectors, host cells, antibodies, and recombinant and synthetic methods for producing said

polypeptides. The invention further relates to diagnostic and therapeutic methods for applying these novel CAN-12, CAN-12v1, and CAN-12v2 polypeptides to the diagnosis, treatment, and/or prevention of various diseases and/or disorders related to these polypeptides, particularly neuro- and musculo-degenerative conditions. The invention further relates to screening methods for identifying agonists and antagonists of the polynucleotides and polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 8 OF 10 USPATFULL on STN
AN 2002:221781 USPATFULL
TI Methods and compositions for producing a neurosalutary effect in a subject
IN Benowitz, Larry I., Newton Square, MA, UNITED STATES
PI US 2002119923 A1 20020829
AI US 2001-872347 A1 20010601 (9)
PRAI US 2000-208778P 20000601 (60)
DT Utility
FS APPLICATION
LREP LAHIVE & COCKFIELD, 28 STATE STREET, BOSTON, MA, 02109
CLMN Number of Claims: 46
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 1372
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods and compositions for producing a neurosalutary effect in a subject, such as modulating neuronal survival and/or regeneration in a subject, are provided. Pharmaceutical and packaged formulations are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 9 OF 10 USPAT2 on STN
AN 2003:166515 USPAT2
TI Polynucleotides encoding novel cysteine proteases of the calpain superfamily, CAN-12v1 and CAN-12v2.
IN Chen, Jian, Princeton, NJ, UNITED STATES
Nelson, Thomas C., Lawrenceville, NJ, UNITED STATES
Vaz, Roy J., North Branch, NJ, UNITED STATES
Duclos, Franck, Washington Crossing, PA, UNITED STATES
PA Bristol-Myers Squibb Company, Princeton, NJ, UNITED STATES (U.S. corporation)
PI US 7186564 B2 20070306
AI US 2002-116519 20020403 (10)
PRAI US 2001-300620P 20010625 (60)
US 2001-296180P 20010606 (60)
US 2001-288768P 20010504 (60)
US 2001-281253P 20010403 (60)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Nashed, Nashaat T.; Assistant Examiner: Moore, William W.
LREP D'Amico, Stephen C.
CLMN Number of Claims: 18
ECL Exemplary Claim: 1
DRWN 27 Drawing Figure(s); 27 Drawing Page(s)
LN.CNT 30048
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides novel polynucleotides encoding CAN-12 polypeptides, fragments and homologues thereof. The present invention also provides polynucleotides encoding variants of CAN-12 polypeptides, CAN-12v1 and CAN-12v2. Also provided are vectors, host cells,

antibodies, and recombinant and synthetic methods for producing said polypeptides. The invention further relates to diagnostic and therapeutic methods for applying these novel CAN-12, CAN-12v1, and CAN-12v2 polypeptides to the diagnosis, treatment, and/or prevention of various diseases and/or disorders related to these polypeptides, particularly neuro- and musculo-degenerative conditions. The invention further relates to screening methods for identifying agonists and antagonists of the polynucleotides and polypeptides of the present invention.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 10 OF 10 WPINDEX COPYRIGHT 2007 THE THOMSON CORP on STN
AN 2004-316013 [29] WPINDEX
DNC C2004-119849 [29]
TI Use of hexose (e.g. D-mannose) to treat/alleviate neurological disorders such as traumatic brain injury, stroke, cerebral aneurysm, Parkinson's disease, amyotrophic lateral sclerosis and Alzheimer's disease
DC B03; B04
IN BENOWITZ L I
PA (CHIL-N) CHILDRENS MEDICAL CENT
CYC 104
PIA WO 2004028468 A2 20040408 (200429)* EN 59[9]
AU 2003272728 A1 20040419 (200462) EN
EP 1542702 A2 20050622 (200541) EN
US 20050256059 A1 20051117 (200576) EN
JP 2006503847 W 20060202 (200611) JA 35
AU 2003272728 A8 20051103 (200629) EN
CN 1703227 A 20051130 (200636) ZH
ADT WO 2004028468 A2 WO 2003-US30466 20030925; US 20050256059 A1 Provisional US 2002-414063P 20020927; AU 2003272728 A1 AU 2003-272728 20030925; AU 2003272728 A8 AU 2003-272728 20030925; EP 1542702 A2 EP 2003-754929 20030925; EP 1542702 A2 WO 2003-US30466 20030925; US 20050256059 A1 WO 2003-US30466 20030925; JP 2006503847 W WO 2003-US30466 20030925; JP 2006503847 W JP 2004-540004 20030925; US 20050256059 A1 US 2005-528685 20050718; CN 1703227 A CN 2003-825428 20030925
FDT AU 2003272728 A1 Based on WO 2004028468 A; EP 1542702 A2 Based on WO 2004028468 A; JP 2006503847 W Based on WO 2004028468 A; AU 2003272728 A8 Based on WO 2004028468 A
PRAI US 2002-414063P 20020927
US 2005-528685 20050718
AN 2004-316013 [29] WPINDEX
AB WO 2004028468 A2 UPAB: 20060203
NOVELTY - Treatment of a neurological disorder comprises the administration of a hexose (I).

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:

(1) an article of manufacture that comprises a pharmaceutical agent (A) (comprising D-mannose) contained within a packaging material which comprises a label indicating that (A) may be administered together with a carrier for a sufficient term at an effective dose to treat a neurological disorder; and

(2) a formulation comprising D-mannose, a cyclic adenosine monophosphate (cAMP) modulator and a carrier.

ACTIVITY - Neuroprotective; Vulnerary; Cerebroprotective; Vasotropic; Antiparkinsonian; Nootropic; CNS-Gen.; Anticonvulsant; Neuroleptic; Muscular-Gen.; Relaxant; Antiinflammatory; Ophthalmological.

The axon-promoting effects of hexose sugars and related compounds were tested on retinal ganglion cells in culture. (I) exhibited a median effective dosage value of approximately 10 microM.

MECHANISM OF ACTION - None given in the source material.

USE - Treatment with (I) reverses neuronal damage and treats/alleviates neurological disorders (preferably

traumatic brain injury, stroke, cerebral aneurysm, Parkinson's disease, amyotrophic lateral sclerosis, Alzheimer's disease, diffuse cerebral cortical atrophy, Lewy-body dementia, Pick disease, mesolimbocortical dementia, thalamic degeneration, Huntington's chorea, cortical-striatal-spinal degeneration, cortical-basal ganglionic degeneration, cerebrocerebellar degeneration, familial dementia with spastic paraparesis, polyglucosan body disease, Shy-Drager syndrome, olivopontocerebellar atrophy, progressive supranuclear palsy, dystonia musculorum deformans, Hallervorden-Spatz disease, Meige syndrome, familial tremors, Gilles de la Tourette syndrome, acanthocytic chorea, Friedreich ataxia, Holmes familial cortical cerebellar atrophy, Gerstmann-Straussler-Scheinker disease, progressive spinal muscular atrophy, progressive balbar palsy, primary lateral sclerosis, hereditary muscular atrophy, spastic paraplegia , peroneal muscular atrophy, hypertrophic interstitial polyneuropathy, heredopathia atactica polyneuritiformis, optic neuropathy, ophthalmoplegia and, particularly, spinal cord injury (characterized by monoplegia, diplegia, paraplegia, hemiplegia and quadriplegia), retinal damage (resulting from macular degeneration) or optic nerve damage (resulting from glaucoma) (all claimed).

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FULL ESTIMATED COST                                   238.83     239.88

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FILE LAST UPDATED: 1 Nov 2007  (20071101/ED)

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=> s benowitz larry I?/AU
L18          69 BENOWITZ LARRY I?/AU

=> s l18 and neurological
           4127 NEUROLOGICAL
              9 NEUROLOGICALS
           4136 NEUROLOGICAL
                  (NEUROLOGICAL OR NEUROLOGICALS)
          27817 NEUROL
          29179 NEUROLOGICAL
                  (NEUROLOGICAL OR NEUROL)
L19           5 L18 AND NEUROLOGICAL
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=> dis l19 1-5 bib abs

L19  ANSWER 1 OF 5  CAPLUS  COPYRIGHT 2007 ACS on STN
AN   2004:290472  CAPLUS
DN   140:264527
TI   Methods and compositions for treatment of neurological disorder
IN   Benowitz, Larry I.
PA   Children's Medical Center Corporation, USA
SO   PCT Int. Appl., 59 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.         DATE
     --------------      ----   --------    --------------------    --------
PI   WO 2004028468        A2    20040408    WO 2003-US30466         20030925
     WO 2004028468        A3    20041021
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM,
             PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
             TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
             FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
             BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     CA 2499170           A1    20040408    CA 2003-2499170         20030925
     AU 2003272728        A1    20040419    AU 2003-272728          20030925
     EP 1542702           A2    20050622    EP 2003-754929          20030925
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
     CN 1703227           A     20051130    CN 2003-825428          20030925
     JP 2006503847        T     20060202    JP 2004-540004          20030925
     US 2005256059        A1    20051117    US 2005-528685          20050718
PRAI US 2002-414063P      P     20020927
     WO 2003-US30466      W     20030925
AB   The invention provides methods and compns. for producing a neurosalutary
     effect in a subject useful for the treatment of neurol.
     disorders, including retinal and optic nerve damage, in a subject in need
     thereof.  The method includes administration to a subject a
     therapeutically effective amount of a hexose, such as mannose.

L19  ANSWER 2 OF 5  CAPLUS  COPYRIGHT 2007 ACS on STN
AN   2002:184938  CAPLUS
DN   136:241683
TI   Sequence of a novel bovine N-kinase and therapeutic uses for producing a
     neurosalutary effect
IN   Benowitz, Larry I.
PA   Children's Medical Center Corporation, USA
SO   PCT Int. Appl., 42 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 2
     PATENT NO.          KIND   DATE        APPLICATION NO.         DATE
     --------------      ----   --------    --------------------    --------
PI   WO 2002020056        A2    20020314    WO 2001-US27691         20010907
     WO 2002020056        A3    20030313
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL,
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PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

	US 7172871	B1	20070206	US 2000-656915	20000907
	CA 2419456	A1	20020314	CA 2001-2419456	20010907
	AU 200187118	A	20020322	AU 2001-87118	20010907
	EP 1315514	A2	20030604	EP 2001-966619	20010907
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	JP 2004523470	T	20040805	JP 2002-524539	20010907
	US 2007231376	A1	20071004	US 2006-640811	20061218
PRAI	US 2000-656915	A	20000907		
	WO 2001-US27691	W	20010907		

AB The invention provides protein sequence of a novel bovine N-kinase, which is an isoform of protein kinase MST-3, and methods for modulating its activity to produce a neurosalutary effects. These methods generally involve administering to subject a therapeutically effective amount of a compound that modulates the activity of N-kinase, or analog thereof. Pharmaceutical and packaged formulations including the compds. of the invention, e.g., compds. that modulate the activity of N-kinase, are also provided.

L19 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:72155 CAPLUS
DN 136:113174
TI Neurotrophic factors present in Schwann cell conditioned media, compositions containing the factors, and methods useful in treating neurological conditions
IN Benowitz, Larry I.; Irwin, Carleen A.; Jackson, Paul
PA Children's Medical Center Corporation, USA
SO PCT Int. Appl., 50 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002006341	A1	20020124	WO 2001-US22315	20010716
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
PRAI	US 2000-616287	A	20000714		

AB The invention provides neurotrophic factors, present in Schwann cell conditioned media, compns. containing the factors, and methods useful in treating neurol. conditions. The neurotrophic factor is mammalian AF-1 (axogenesis factor 1). The neurotrophic factors can also be administered with a macrophage-derived factor or a cAMP modulator.
RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L19 ANSWER 4 OF 5 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:885803 CAPLUS
DN 136:684
TI Methods and compositions for producing a neurosalutary effect in a subject
IN Benowitz, Larry I.
PA Children's Medical Center Corp., USA

SO PCT Int. Appl., 35 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001091783	A2	20011206	WO 2001-US17895	20010601
	WO 2001091783	A3	20020711		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2411666	A1	20011206	CA 2001-2411666	20010601
	EP 1289540	A2	20030312	EP 2001-946052	20010601
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	JP 2003534385	T	20031118	JP 2001-587797	20010601
	US 2005059594	A1	20050317	US 2004-894351	20040719
PRAI	US 2000-208778P	P	20000601		
	US 2001-872347	B1	20010601		
	WO 2001-US17895	W	20010601		

AB Methods and compns. for producing a neurosalutary effect in a subject, such as modulating neuronal survival and/or regeneration in a subject, are provided. The present invention provides methods and compns. for producing a neurosalutary effect in a subject with a neurol. condition; such effects include promoting neuronal survival, axonal outgrowth, neuronal regeneration or normalized neurol. function in a subject. Pharmaceutical and packaged formulations are also provided.

L19 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1999:66625 CAPLUS
DN 130:306436
TI Tacrolimus (FK506) increases neuronal expression of GAP-43 and improves functional recovery after spinal cord injury in rats
AU Madsen, Joseph R.; MacDonald, Paul; Irwin, Nina; Goldberg, David E.; Yao, Gui-Lan; Meiri, Karina F.; Rimm, Ilonna J.; Stieg, Philip E.; Benowitz, Larry I.
CS Department of Neurosurgery, Children's Hospital, Boston, MA, 02115, USA
SO Experimental Neurology (1998), 154(2), 673-683
CODEN: EXNEAC; ISSN: 0014-4886
PB Academic Press
DT Journal
LA English
AB Tacrolimus (FK506), a widely used immunosuppressant drug, has neurite-promoting activity in cultured PC12 cells and peripheral neurons. The present study investigated whether tacrolimus affects the expression of the neuronal growth-associated protein, GAP-43, as well as functional recovery after photothrombotic spinal cord injury in the rat. In injured animals receiving tacrolimus, the number of neurons expressing GAP-43 mRNA and protein approx. doubled compared to that in injured animals receiving vehicle alone. This increase in GAP-43-pos. cells was paralleled by a significant improvement in neurol. function evaluated by open-field and inclined plane tests. Another FKBP-12 ligand (V-10,367) had similar effects on GAP-43 expression and functional outcome, indicating that the observed effects of tacrolimus do not involve inhibition of the phosphatase calcineurin. Thus, tacrolimus, a drug which is already approved for use in humans, as well as other FKBP-12 ligands which do not inhibit calcineurin, could potentially enhance functional outcome after

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     CNS injury in humans.  (c) 1998 Academic Press.
RE.CNT  43     THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD
               ALL CITATIONS AVAILABLE IN THE RE FORMAT


=> dis hist

     (FILE 'HOME' ENTERED AT 15:23:35 ON 02 NOV 2007)

     FILE 'APOLLIT, MEDLINE, BIOSIS, EMBASE, BABS, CAPLUS, CBNB, CIN,
     COMPENDEX, DISSABS, EMA, IFIPAT, NTIS, PASCAL, PROMT, RAPRA, SCISEARCH,
     TEXTILETECH, USPATFULL, USPATOLD, USPAT2, WPIFV, WPINDEX, WSCA,
     WTEXTILES' ENTERED AT 15:26:19 ON 02 NOV 2007
L1        192059 S NEUROLOGICAL AND DISORDER
L2          3665 S L1 AND (MANNOSE OR GULOSE OR GLUCOSE-6-PHOSPHATE)
L3          1090 S L2 AND (CAMP AND MODULATOR)
L4            11 S L3 AND ONCOMODULIN
L5          1062 S L3 AND (STROKE OR ANEURISM OR SPINAL OR PARKINSON OR SCLEROS
L6           832 S L5 AND MACROPHAGE
L7           832 S L6 AND FACTOR
L8           661 S L7 AND TGF
L9           620 S L8 AND ALZHEIMER
L10           10 S L9 AND ONCOMODULIN
L11          619 S L9 AND NEURON?
L12          408 S L8 AND GLAUCOMA
L13          368 S L12 AND INTRAOCULAR
L14          367 S L13 AND INJECT?
L15         6805 S L1 AND RETINA?
L16          358 S L14 AND RETINA?
L17            5 S L10 AND MACULAR

     FILE 'CAPLUS' ENTERED AT 15:43:13 ON 02 NOV 2007
L18           69 S BENOWITZ LARRY I?/AU
L19            5 S L18 AND NEUROLOGICAL

=> s l1 and mannose
          41511 MANNOSE
            233 MANNOSES
          41562 MANNOSE
                (MANNOSE OR MANNOSES)
L20           24 L1 AND MANNOSE

=> dis l20 1-24 bib abs

L20  ANSWER 1 OF 24  CAPLUS  COPYRIGHT 2007 ACS on STN
AN   2007:201705  CAPLUS
DN   146:267946
TI   Gene disruptions in murine genes for characterization of their functions
     and the functions of their human orthologs
IN   Byers-Horner, Allison Anne; Combs, Katherin; Culbertson, Ling Ling;
     Delmas-Mata, Juan; Desauvage, Frederic; Fan, Liangfen; Frantz, Gretchen;
     Green, Leslie Jane; Massey, Erin Marie; McLain, Dina Rebecca; Montgomery,
     Chuck; Payne, Bobby Joe; Peale, Franklin, Jr.; Phillips, Heidi; Rohrer,
     Michelle; Shi, Zheng-Zheng; Sparks, Mary Jean; Stala, Joy; Tang, Tracy
     Tzu-Ling; Vogel, Peter; Wang, Ching-Yun; Willis-Sevaux, Tracy Ellen;
     Xiong, Wen
PA   Genentech, Inc., USA; Lexicon Genetics Incorporated
SO   PCT Int. Appl., 591pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.        DATE
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PI WO 2007021423 A2 20070222 WO 2006-US27777 20060718
WO 2007021423 A3 20070628
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA
PRAI US 2005-708312P P 20050815
AB The present invention relates to transgenic animals, as well as compns. and methods relating to the characterization of gene function. Specifically, the present invention provides transgenic mice comprising disruptions in 53 genes, thereby identifying the phenotyping functions of these mouse genes and their human orthologs. Such in vivo studies and characterizations may provide valuable identification and discovery of therapeutics and/or treatments useful in the prevention, amelioration or correction of diseases or dysfunctions associated with gene disruptions such as neurol. disorders; cardiovascular, endothelial or angiogenic disorders; eye abnormalities; immunol. disorders; oncol. disorders; bone metabolic abnormalities or disorders; lipid metabolic disorders; or developmental abnormalities.

L20 ANSWER 2 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:1312589 CAPLUS
DN 146:56558
TI Identification of novel genes and proteins, gene disruption in transgenic animals, and uses in drug screening and treating human disorders
IN Byers-Horner, Allison Anne; Combs, Katherin; Culbertson, Ling Ling; Desauvage, Frederic; Ding, Zhiyong; Edwards, Joel; Girgis, Rosemary; Junge, Harald; Junutula, Jagath Reddy; Massey, Erin Marie; Mclain, Dina Rebecca; Montgomery, Chuck; Payne, Bobby Joe; Phillips, Heidi; Qian, Ni Nancy; Rangel, Carolina; Shi, Zheng-Zheng; Sparks, Mary Jean; Stala, Joy; Vogel, Peter; Willis-Sevaux, Tracy Ellen; Ye, Weilan
PA Genentech, Inc., USA; Lexicon Genetics Incorporated
SO PCT Int. Appl., 746pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006132788	A2	20061214	WO 2006-US19651	20060518
WO 2006132788	A3	20070726		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

PRAI US 2005-687900P P 20050606
US 2006-780262P P 20060307

AB Seventy-one novel proteins are identified by extracellular domain homol. screening and isolation of cDNA clones from human tissues by amylase screening and signal algorithm anal. Biol. functions are identified by disruptions in the genes encoding the proteins, tissue expression profiling, and microarray anal. in cancerous tissues. Such in vivo studies and characterizations may provide valuable identification and discovery of therapeutics and/or treatments useful in the prevention, amelioration or correction of diseases or dysfunctions associated with gene disruptions such as neurol. disorders; cardiovascular, endothelial or angiogenic disorders; eye abnormalities; immunol. disorders; oncol. disorders; bone metabolic abnormalities or disorders; lipid metabolic disorders; or developmental abnormalities.

L20 ANSWER 3 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:764397 CAPLUS
DN 145:267441
TI Targeted disruption of the mouse phosphomannomutase 2 gene causes early embryonic lethality
AU Thiel, Christian; Luebke, Torben; Matthijs, Gert; von Figura, Kurt; Koerner, Christian
CS Universitaetskinderklinik Heidelberg, Abteilung I, Friedrich Karls Universitaet Heidelberg, Heidelberg, 69120, Germany
SO Molecular and Cellular Biology (2006), 26(15), 5615-5620
CODEN: MCEBD4; ISSN: 0270-7306
PB American Society for Microbiology
DT Journal
LA English
AB Mutations in the cytosolic enzyme phosphomannomutase 2 (PMM2), which catalyzes the conversion of mannose-6-phosphate to mannose-1-phosphate, cause the most common form of congenital disorders of glycosylation, termed CDG-Ia. It is an inherited multi-systemic disease with severe neurol. impairment. To study the pathophysiol. of CDG-Ia and to investigate possible therapeutic approaches, we generated a mouse model for CDG-Ia by targeted disruption of the Pmm2 gene. Heterozygous mutant mice appeared normal in development, gross anatomy, and fertility. In contrast, embryos homozygous for the Pmm2-null allele were recovered in embryonic development at days 2.5 to 3.5. These results indicate that Pmm2 is essential for early development of mice. Mating expts. of heterozygous mice with wild-type mice could further show that transmission of the female Pmm2-null allele is impaired.
RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 4 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:555308 CAPLUS
DN 143:169025
TI Over-expression of human lysosomal α-mannosidase in mouse embryonic stem cells
AU Robinson, A. J.; Crawley, A. C.; Hopwood, J. J.
CS Lysosomal Diseases Research Unit, Department of Genetic Medicine, Women's and Children's Hospital, North Adelaide, 5006, Australia
SO Molecular Genetics and Metabolism (2005), 85(3), 203-212
CODEN: MGMEFF; ISSN: 1096-7192
PB Elsevier
DT Journal
LA English
AB α-Mannosidosis is a lysosomal storage disorder characterized by the lysosomal accumulation of mannose-containing oligosaccharides and a range of pathol. consequences, caused by a deficiency of the lysosomal enzyme α-mannosidase. One of the major features of α-mannosidosis is progressive neurol. decline, for which there is no safe and effective treatment. Implantation of stem

cells into the central nervous system has been proposed as a potential therapy for these disorders. We report the construction and characterization of mouse embryonic stem cell lines for the sustained over-expression of recombinant human lysosomal α-mannosidase (rhαM). Two vectors (involving recombinant human α-mannosidase expression driven by either the chicken β-actin promoter/CMV enhancer or by the elongation factor 1-α promoter) were constructed and used to transfect mouse D3 embryonic stem cells. Selected clonal cell lines were isolated and tested to evaluate their expression of recombinant human α-mannosidase. Stem cell clones transfected with the chicken β-actin promoter/CMV enhancer maintained rhαM expression levels throughout differentiation. This expression was not markedly elevated above background. In contrast, the vector incorporating the elongation factor 1-α promoter facilitated substantial over-expression of α-mannosidase when analyzed out to 21 days of differentiation in stably transfected cell lines. The highest expressing cell line was found to qual. retain a similar differentiation potential to untransfected cells, and to secrete α-mannosidase that could mediate a reduction in the level of oligosaccharides stored by human α-mannosidosis skin fibroblasts. These results suggest potential for the use of this cell line for investigation of a stem cell therapy approach to treat α-mannosidosis.

RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 5 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:379205 CAPLUS
DN 143:76224
TI Analysis of Glycosylation in CDG-Ia Fibroblasts by Fluorophore-assisted Carbohydrate Electrophoresis: implications for extracellular glucose and intracellular mannose 6-phosphate
AU Gao, Ningguo; Shang, Jie; Lehrman, Mark A.
CS Department of Pharmacology, University of Texas-Southwestern Medical Center, Dallas, TX, 75390, USA
SO Journal of Biological Chemistry (2005), 280(18), 17901-17909
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
AB Phosphomannomutase (PMM) deficiency causes congenital disorder of glycosylation (CDG)-Ia, a broad spectrum disorder with developmental and neurol. abnormalities. PMM converts mannose 6-phosphate (M6P) to mannose-1-phosphate, a precursor of GDP-mannose used to make Glc3Man9GlcNAc2-P-P-dolichol (lipid-linked oligosaccharide; LLO). LLO, in turn, is the donor substrate of oligosaccharyltransferase for protein N-linked glycosylation. Hepatically produced N-linked glycoproteins in CDG-Ia blood are hypoglycosylated. Upon labeling with [3H]mannose, CDG-Ia fibroblasts have been widely reported to accumulate [3H]LLO intermediates. Since these are thought to be poor oligosaccharyltransferase substrates, LLO intermediate accumulation has been the prevailing explanation for hypoglycosylation in patients. However, this is discordant with sporadic reports of specific glycoproteins (detected with antibodies) from CDG-Ia fibroblasts being fully glycosylated. Here, fluorophore-assisted carbohydrate electrophoresis (FACE, a nonradioactive technique) was used to analyze steady-state LLO compns. in CDG-Ia fibroblasts. FACE revealed that low glucose conditions accounted for previous observations of accumulated [3H]LLO intermediates. Addnl. FACE expts. demonstrated abundant Glc3Man9GlcNAc2-P-P-dolichol, without hypoglycosylation, CDG-Ia fibroblasts grown with physiol. glucose. This suggested a "missing link" to explain hypoglycosylation in CDG-Ia patients. Because of the possibility of its accumulation, the effects of M6P on glycosylation were explored in vitro. Surprisingly, M6P was a specific activator for cleavage of Glc3Man9GlcNAc2-P-P-dolichol. This led to futile cycling the

LLO pathway, exacerbated by GDP-mannose/PMM deficiency. The possibilities that M6P may accumulate in hepatocytes and that M6P-stimulated LLO cleavage may account for both hypoglycosylation and the clin. failure of dietary mannose therapy with CDG-Ia patients are discussed.

RE.CNT 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 6 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:128705 CAPLUS
DN 142:328763
TI Gene therapy of metachromatic leukodystrophy
AU Matzner, Ulrich; Gieselmann, Volkmar
CS Institut fuer Physiologische Chemie, Rheinische Friedrich-Wilhelms-Universitaet, Bonn, D-53115, Germany
SO Expert Opinion on Biological Therapy (2005), 5(1), 55-65
CODEN: EOBTA2; ISSN: 1471-2598
PB Ashley Publications Ltd.
DT Journal; General Review
LA English
AB A review. Metachromatic leukodystrophy (MLD) is a lysosomal storage disease that is caused by a deficiency of arylsulfatase A (ASA). The deficiency results in the intralysosomal accumulation of the acidic sphingolipid 3-O-sulfogalactosylceramide (sulfatide). Patients suffer from progressive demyelination and die from multiple neurol. deficits. Curative treatment is not available. ASA bears mannose 6-phosphate residues which function as recognition markers in endosome/lysosome-specific targeting pathways. The endocytic targeting route can be exploited to deliver exogeneous ASA to the lysosomes of ASA-deficient cells. ASA knockout mice, which develop a disorder related to MLD, have therefore been treated by ex vivo and in vivo gene therapy. Following transplantation of bone marrow cells overexpressing ASA from a retroviral vector, donor-type cells secrete ASA, which is endocytosed by recipient cells. The enzyme transfer results in the metabolic cross-correction of recipient cells and the improvement of biochem., histol. and clin. parameters. For the transfer of the ASA cDNA to non-dividing cells, adenovirus, adenoassocd. virus and lentivirus vectors have been constructed. Such vectors might be particularly advantageous for direct ASA gene delivery to the brain, which is the main site of disease in MLD.

RE.CNT 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 7 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:974900 CAPLUS
DN 142:275867
TI Purification and characterization of recombinant murine sulfamidase
AU Gliddon, B. L.; Yogalingam, G.; Hopwood, J. J.
CS Department of Genetic Medicine, Lysosomal Diseases Research Unit, Women's and Children's Hospital, North Adelaide, 5006, Australia
SO Molecular Genetics and Metabolism (2004), 83(3), 239-245
CODEN: MGMEFF; ISSN: 1096-7192
PB Elsevier
DT Journal
LA English
AB Mucopolysaccharidosis type IIIA (MPS IIIA) is a lysosomal storage disorder caused by a deficiency in the lysosomal enzyme sulfamidase, which is required for the degradation of heparan sulfate. The disease is characterized by neurol. dysfunction but relatively mild somatic manifestations. A naturally occurring mouse model to MPS IIIA exhibits a similar disease progression to that observed in patients. Disease in the mice results from a base substitution at codon 31 in the sulfamidase gene, altering an aspartic acid to an asparagine (D31N). This aspartic 31 is involved in binding of the divalent metal ion needed for

catalytic function, and as such reduces the specific activity of the enzyme to about 3% of that of wild-type. The mutant protein has decreased stability and shows increased degradation over a 24 h chase period when compared to wild-type mouse sulfamidase. Mouse sulfamidase that was purified using a two-step ion exchange procedure was shown to have similar kinetic properties to that of purified human sulfamidase. Recombinant murine sulfamidase was able to correct the storage phenotype of MPS IIIA fibroblasts after endocytosis via the mannose-6-phosphate receptor.

RE.CNT 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 8 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:452933 CAPLUS
DN 141:37230
TI Nuclear receptors as diagnostic and risk markers for disease and as targets for therapy
IN Gaitanaris, George A.; Bergmann, John E.; Gracerov, Alexander; Hohmann, John; Li, Fusheng; Madisen, Linda; Mcilwain, Kellie L.; Pavlova, Maria N.; Vassilatis, Demetri; Zeng, Hongkui
PA Nura, Inc., USA
SO PCT Int. Appl., 508 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004045369	A2	20040603	WO 2003-US36229	20031112
	WO 2004045369	A3	20070301		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2003295500	A1	20040615	AU 2003-295500	20031112
PRAI	US 2002-426305P	P	20021114		
	WO 2003-US36229	W	20031112		

AB Methods of using nuclear receptors as diagnostic markers for disease and for increased risk of disease and in the development of therapeutics for treatment of such diseases are described. The proteins and the genes encoding them may be used in diagnosis. Transgenic animals carrying the human genes for these receptors may be used in screening for effectors. The invention also provides methods for identifying compds. (e.g., agonists or antagonists) using the nuclear receptor polypeptides and polynucleotides of the invention, and for treating conditions associated with nuclear receptor dysfunction with the nuclear receptor polypeptides, polynucleotides, or identified compds. The invention also provides diagnostic assays for detecting diseases or disorders associated with inappropriate nuclear receptor activity or levels.

L20 ANSWER 9 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:290472 CAPLUS
DN 140:264527
TI Methods and compositions for treatment of neurological disorder
IN Benowitz, Larry I.
PA Children's Medical Center Corporation, USA
SO PCT Int. Appl., 59 pp.

CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004028468	A2	20040408	WO 2003-US30466	20030925
WO 2004028468	A3	20041021		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2499170	A1	20040408	CA 2003-2499170	20030925
AU 2003272728	A1	20040419	AU 2003-272728	20030925
EP 1542702	A2	20050622	EP 2003-754929	20030925
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
CN 1703227	A	20051130	CN 2003-825428	20030925
JP 2006503847	T	20060202	JP 2004-540004	20030925
US 2005256059	A1	20051117	US 2005-528685	20050718
PRAI US 2002-414063P	P	20020927		
WO 2003-US30466	W	20030925		

AB The invention provides methods and compns. for producing a neurosalutary effect in a subject useful for the treatment of neurol. disorders, including retinal and optic nerve damage, in a subject in need thereof. The method includes administration to a subject a therapeutically effective amount of a hexose, such as mannose.

L20 ANSWER 10 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:50529 CAPLUS
DN 140:233949
TI In vitro characterization of genetically modified embryonic stem cells as a therapy for murine mucopolysaccharidosis type IIIA
AU Lau, Adeline A.; Hemsley, Kim M.; Meedeniya, Adrian; Hopwood, John J.
CS Department of Genetic Medicine, Lysosomal Diseases Research Unit, Women's and Children's Hospital, North Adelaide, 5006, Australia
SO Molecular Genetics and Metabolism (2004), 81(2), 86-95
CODEN: MGMEFF; ISSN: 1096-7192
PB Elsevier Science
DT Journal
LA English
AB The mucopolysaccharidoses (MPS) are lysosomal storage disorders resulting from the impaired catabolism of glycosaminoglycans (GAG). MPS type IIIA patients have dysfunctional sulfamidase enzyme leading to lysosomal storage of the GAG heparan sulfate, severe neurol. symptoms including regression in learning, behavioral abnormalities, and premature death. The authors have engineered mouse D3 embryonic stem (ES) cells to over-express recombinant human sulfamidase. Human sulfamidase was correctly folded and secreted 2 h post-labeling as determined by immunopptn. and SDS-PAGE anal. of transfected ES cells. Secreted human sulfamidase present in conditioned ES cell media was able to be taken up via mannose-6-phosphate-mediated endocytosis and restored sulfamidase enzyme activity in human MPS IIIA fibroblast cell lines. ES cells underwent directed differentiation to neural precursor populations and were capable of sustained human sulfamidase over-expression at all stages. Addnl., transfected and control cells were proliferative (Ki67+) and expressed several neural markers (nestin, MAP-2, and NF160) as determined by immunofluorescence. These findings suggest the possibility of ES

cell-based therapy for the treatment of neurol. pathol. of MPS IIIA.
RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 11 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:18723 CAPLUS
DN 140:71049
TI Novel compositions and methods for treating neurological disorders and associated gastrointestinal conditions
IN Brudnak, Mark A.
PA MAK Wood, Inc., USA
SO U.S. Pat. Appl. Publ., 13 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2004005304	A1	20040108	US 2002-191385	20020708
PRAI US 2002-191385		20020708		

AB The present invention provides therapeutic compns. and methods for treating to neurol. disorders and associated gastrointestinal conditions using enhancer mols. These enhancer mols. comprise therapeutically effective amts. of metals, amino acids, polypeptides, saccharides, probiotics, and combinations thereof to enhance expression of genes, and/or enzymic activity of gastrointestinal proteins.

L20 ANSWER 12 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:806989 CAPLUS
DN 138:331147
TI Uptake of recombinant iduronate-2-sulfatase into neuronal and glial cells in vitro
AU Daniele, A.; Tomanin, R.; Villani, G. R. D.; Zacchello, F.; Scarpa, M.; Di Natale, P.
CS Medical School, Department of Biochemistry and Medical Biotechnologies, University of Naples Federico II, Naples, Italy
SO Biochimica et Biophysica Acta, Molecular Basis of Disease (2002), 1588(3), 203-209
CODEN: BBADEX; ISSN: 0925-4439
PB Elsevier B.V.
DT Journal
LA English
AB Mucopolysaccharidosis type II (MPS II, Hunter syndrome) is a congenital storage disorder resulting from mutations on the iduronate-2-sulfatase (IDS) gene. The disease shows variable clin. phenotypes from severe to mild with progressive neurol. dysfunction. The therapeutic options for treatment of MPS II are limited and currently no specific therapies are available; the problem is further compounded by difficulties in delivering therapeutic agents to the central nervous system (CNS). In this work, as a potential treatment for this disease, the transfer of the recombinant IDS enzyme into brain cells has been studied in vitro. Two different approaches to obtain recombinant IDS have been utilized: production of the recombinant enzyme by a transfected human clone (Bosc 23 cells); production of the recombinant enzyme by adenoviral transduction of neuronal (SK-N-BE) or glial (C6) cells. Our data indicate that the transfected as well as the infected cells produce a large amount of the IDS enzyme, which is efficiently endocytosed into neuronal and glial cells through the mannose 6-phosphate (M6P) receptor system. Somatic gene therapy appears therefore to be suitable to correct IDS deficiency in brain cells.
RE.CNT 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 13 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2002:556871 CAPLUS
DN 137:245977
TI Truncated, inactive N-acetylglucosaminyltransferase III (GlcNAc-TIII) induces neurological and other traits absent in mice that lack GlcNAc-TIII
AU Bhattacharyya, Riddhi; Bhaumik, Mantu; Raju, T. Shantha; Stanley, Pamela
CS Department of Cell Biology, Albert Einstein College of Medicine, New York, NY, 10461, USA
SO Journal of Biological Chemistry (2002), 277(29), 26300-26309
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
AB N-Acetylglucosaminyltransferase III (GlcNAc-TIII), the product of the Mgat3 gene, transfers the bisecting GlcNAc to the core mannose of complex N-glycans. The addition of this residue is regulated during development and has functional consequences for receptor signaling, cell adhesion, and tumor progression. Mice homozygous for a null mutation at the Mgat3 locus (Mgat3Δ) or for a targeted mutation in the Mgat3 gene (previously called Mgat3neo, but herein renamed Mgat3T37 because the allele generates inactive GlcNAc-TIII of .apprx.37 kDa) were found to exhibit retarded progression of liver tumors. Matrix-assisted laser desorption/ionization time-of-flight mass spectrometry of neutral N-glycans from kidneys revealed no significant differences, and both mutants showed the expected lack of N-glycan species with an addnl. GlcNAc. However, the two mutants differed in several biol. traits. Mgat3T37/T37 homozygotes in a mixed or 129SvJ background were retarded in growth rate and exhibited an altered leg clasp reflex, an altered gait, and defective nursing behavior. Pups abandoned by Mgat3T37/T37 mothers were rescued by wild-type foster mothers. None of these Mgat3T37/T37 traits were exhibited by Mgat3Δ/Δ mice or by heterozygous mice carrying the Mgat3T37 mutation. Similarly, no dominant-neg. effect was observed in Chinese hamster ovary cells expressing truncated GlcNAc-TIII in the presence of wild-type GlcNAc-TIII. However, compound heterozygotes carrying both the Mgat3T37 and Mgat3Δ mutations exhibited a marked leg clasp reflex, indicating that in the absence of wild-type GlcNAc-TIII, truncated GlcNAc-TIII causes this phenotype. The Mgat3 gene was expressed in brain at embryonic day 10.5 and thereafter and in neurons of adult cerebellum. The mutant Mgat3 gene was also highly expressed in Mgat3T37/T37 brain. This may be the basis of the unexpected neurol. phenotype induced by truncated, inactive GlcNAc-TIII in the mouse.
RE.CNT 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 14 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:713364 CAPLUS
DN 135:267271
TI Probucol-related thioketals and thioethers for inhibiting the expression of VCAM-1, preparation, and therapeutic use
IN Meng, Charles Q.; Hoong, Lee K.; Somers, Patricia K.
PA Atherogenics, Inc., USA
SO PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 4

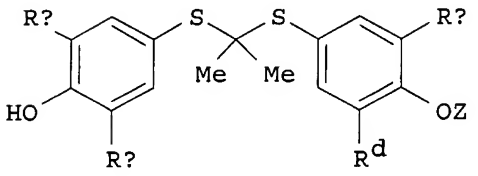
	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001070757	A2	20010927	WO 2001-US9049	20010321
	WO 2001070757	A3	20020314		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM,

HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS,
LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO,
RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ,
VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

CA 2403823	A1	20010927	CA 2001-2403823		20010321
EP 1289944	A2	20030312	EP 2001-920617		20010321
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR					
JP 2003528109	T	20030924	JP 2001-568958		20010321
AU 2001247651	B2	20070104	AU 2001-247651		20010321
AU 2002300328	A1	20021219	AU 2002-300328		20020730
PRAI US 2000-191046P	P	20000321			
AU 1998-74851	A3	19980514			
WO 2001-US9049	W	20010321			

OS MARPAT 135:267271
GI



I

AB Probucol-related thioketals and thioethers are provided that inhibit the expression of VCAM-1, and which can be used in the treatment of VCAM-1-mediated diseases, including inflammatory disorders, cardiovascular diseases, ocular diseases, autoimmune diseases, neurol. disorders, and cancer. Compds. of the invention include I [Ra-Rd = H, (un)substituted alkyl, (un)substituted aryl, etc.; Z = (un)substituted carbohydrate, (un)substituted alditol, (un)substituted C1-10 alkyl terminated by sulfonic acid, etc.]. The compds. also can be used to treat hyperlipidemia and/or hypercholesterolemia. Compound preparation is described.

L20 ANSWER 15 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:337959 CAPLUS
DN 135:151146
TI Functional Analysis of Novel Mutations in a Congenital Disorder of Glycosylation Ia Patient with Mixed Asian Ancestry
AU Westphal, Vibeke; Enns, Gregory M.; McCracken, Marjorie F.; Freeze, Hudson H.
CS The Burnham Institute, La Jolla, CA, 92037, USA
SO Molecular Genetics and Metabolism (2001), 73(1), 71-76
CODEN: MGMEFF; ISSN: 1096-7192
PB Academic Press
DT Journal
LA English
AB Congenital disorders of glycosylation (CDG) are caused by autosomal recessive mutations in genes affecting N-glycan biosynthesis. Mutations in the PMM2 gene, which encodes the enzyme phosphomannomutase (mannose 6-phosphate ↔ mannose 1-phosphate), give rise to the most common form: CDG-Ia. These patients typically present with dysmorphic features and neurol. abnormalities, cerebellar hypoplasia, ataxia, hypotonia, and coagulopathy, in addition to feeding problems. However, the clin. symptoms vary greatly. The great majority of known CDG-Ia patients are of European descent where the most common mutant alleles originated. This ethnic bias can also be explained by lack

of global awareness of the disorder. Here the authors report an Asian patient with prominent systemic features that the authors diagnosed with CDG-Ia resulting from two new mutations in the PMM2 gene (310C→G resulting in L104V and an intronic mutation IVS1-1G→A). The latter mutation seems to result in lower mRNA levels, and the L104V has been functionally analyzed in a yeast expression system together with known mutations. The Filipino and Cambodian origins of the parents show that CDG-Ia mutations occur in these ethnic groups as well as in Caucasians. (c) 2001 Academic Press.

RE.CNT 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 16 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1999:629354 CAPLUS
DN 132:149756
TI Glycosylation defects corrected by the changes in GDPmannose level
AU Kruszewska, Joanna; Janik, Anna; Lenart, Urszula; Palamarczyk, Grazyna
CS Institute of Biochemistry and Biophysics, Polish Academy of Sciences, Warsaw, 02-106, Pol.
SO Acta Biochimica Polonica (1999), 46(2), 315-324
CODEN: ABPLAF; ISSN: 0001-527X
PB Polish Biochemical Society
DT Journal; General Review
LA English
AB A review with 40 refs. GDPMan is a key substrate in glycoprotein formation. This is especially true for lower eukaryotes where, in addition to the involvement in N-glycan biosynthesis and GPI-anchor formation, GDPMan takes part in the process which is unique for yeast and fungi i.e. O-mannosylation. Several lines of evidence have been presented that the level of GDPMan affects the process occurring in the Golgi compartment i.e. the elongation of outer mannose chain of glycoproteins in Saccharomyces cerevisiae. Results from the authors' laboratory indicate that the availability of GDPMan affects also the early steps of glycoprotein formation ascribed to the endoplasmic reticulum, i.e. assembly of the dolichol-linked oligosaccharide as well as mannosyl-phosphodolichol (MPD) formation. The biochem. basis of carbohydrate deficient glycoprotein syndrome, a severe neurol. disorder related to the GDPMan deficiency, is also discussed.

RE.CNT 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 17 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1999:34996 CAPLUS
DN 130:94486
TI Recombinant effector cells expressing chimeric signalling systems and their use in disease treatment
IN Finney, Helene Margaret; Lawson, Alastair David Griffiths; Weir, Andrew Neil Charles
PA Celltech Therapeutics Limited, UK
SO PCT Int. Appl., 53 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9900494	A2	19990107	WO 1998-GB1842	19980624
	WO 9900494	A3	19990325		

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

	AU 9881210	A	19990119	AU 1998-81210	19980624
	EP 1002073	A2	20000524	EP 1998-930934	19980624
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2002511757	T	20020416	JP 1999-505370	19980624
	US 2003060444	A1	20030327	US 2002-280596	20021024
PRAI	GB 1997-13473	A	19970625		
	WO 1998-GB1842	W	19980624		
	US 2000-446529	B1	20000519		

AB A cell activation process is described in which an effector cell is transformed with DNA coding for a chimeric receptor containing two or more different cytoplasmic signalling components. At least one of the cytoplasmic signalling components is derived from all or part of a tetraspan-transmembrane protein, CD43, CD6, a mannose, IL-7, IL-12 or complement receptor, an integrin-associated protein, or a γ-chain associated with a cytokine receptor. The activated cell may be of use in medicine for example in the treatment of diseases such as cancer. Thus, recombinant Jurkat E6.1 cells producing a chimeric receptor consisting of an anti-CD33 single-chain Fv linked via a spacer containing IgG1 and CD28 domains to the intracellular domain of FcεRI, were stimulated to produce interleukin-12 in the presence of CD33-pos. cells.

L20 ANSWER 18 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1998:233726 CAPLUS
DN 129:15098
TI Carbohydrate-deficient glycoprotein syndrome type Ib: phosphomannose isomerase deficiency and mannose therapy
AU Niehues, Ralf; Hasilik, Martin; Alton, Gordon; Korner, Christian; Schiebe-Sukumar, Marika; Koch, Hans Georg; Zimmer, Klaus-Peter; Wu, Rongrong; Harms, Erik; Reiter, Karl; Von Figura, Kurt; Freeze, Hudson H.; Harms, Hinrich Karsten; Marquardt, Thorsten
CS Klinik und Poliklinik fur Kinderheilkunde, Munster, 48149, Germany
SO Journal of Clinical Investigation (1998), 101(7), 1414-1420
CODEN: JCINAO; ISSN: 0021-9738
PB Rockefeller University Press
DT Journal
LA English
AB Phosphomannose isomerase (PMI) deficiency is the cause of a new type of carbohydrate-deficient glycoprotein syndrome (CDGS). The disorder is caused by mutations in the PMI1 gene. The clin. phenotype is characterized by protein-losing enteropathy, while neurol. manifestations prevailing in other types of CDGS are absent. Using standard diagnostic procedures, the disorder is indistinguishable from CDGS type Ia (phosphomannomutase deficiency). Daily oral mannose administration is a successful therapy for this new type of CDG syndrome classified as CDGS type Ib.
RE.CNT 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 19 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1998:51329 CAPLUS
DN 128:214781
TI Recombinant human sulfamidase: expression, amplification, purification and characterization
AU Bielicki, Julie; Hopwood, John J.; Melville, Elizabeth L.; Anson, Donald S.
CS Lysosomal Diseases Research Unit, Department of Chemical Pathology, Women's and Children's Hospital, North Adelaide, 5006, Australia
SO Biochemical Journal (1998), 329(1), 145-150
CODEN: BIJOAK; ISSN: 0264-6021
PB Portland Press Ltd.

DT Journal
LA English
AB Mucopolysaccharidosis type IIIA (MPS IIIA, Sanfilippo A syndrome) is a lysosomal storage disease that causes a profound neurol. deterioration. The disorder is caused by a deficiency of the lysosomal enzyme sulfamidase which is a requisite for the degradation of heparan sulfate. To facilitate the development of enzyme-replacement strategies for MPS IIIA patients, the authors have constructed a high-level expression system for recombinant human sulfamidase in Chinese hamster ovary (CHO) cells. An expression construct containing a methotrexate-resistant dihydrofolate reductase (DHFR) gene allowed amplification of expression levels from less than 1 mg of sulfamidase per L of culture medium to approx. 15 mg/l. Unlike many cell lines made by gene amplification in DHFR-deficient CHO cells, and utilizing the normal DHFR gene, these cell lines appeared to be stable in the absence of selective pressure. Recombinant human sulfamidase was purified from unamplified and amplified cell lines. The native enzyme was found to be a dimer of 115 kDa. Denaturing and reducing SDS-PAGE revealed a subunit size of 62 kDa. Kinetic anal. demonstrated that the recombinant enzyme had broadly similar kinetic characteristics to sulfamidase purified from liver. Recombinant human sulfamidase was able to correct the storage phenotype of MPS IIIA fibroblasts after endocytosis via the mannose-6-phosphate receptor.
RE.CNT 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 20 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1997:300721 CAPLUS
DN 127:651
TI In vitro correction of iduronate-2-sulfatase deficiency by adenovirus-mediated gene transfer
AU Di Francesco, C.; Cracco, C.; Tomanin, R.; Picci, L.; Ventura, L.; Zacchello, F.; Di Natale, P.; Anson, D. S.; Hopwood, J. J.; Graham, F. L.; Scarpa, M.
CS Dep. Pediatrics and Center Biotechnology CRIBI, Univ. Padova, Italy
SO Gene Therapy (1997), 4(5), 442-448
CODEN: GETHEC; ISSN: 0969-7128
PB Stockton
DT Journal
LA English
AB Hunter syndrome is a lethal lysosomal storage disorder caused by the deficiency of iduronate-2-sulfatase and characterized by severe skeletal and neurol. symptoms. Only symptomatic treatments are available and, although bone marrow transplantation has been suggested, no encouraging results have been obtained so far. Therefore, gene therapy might be a route to be pursued for treatment of the disease. In this respect, one major goal to achieve is the generation of an overexpressing vector able to correct, in particular, central nervous system (CNS) cells. Adenoviruses have been shown to infect CNS cells efficiently with minor or even absent immunol. response. We describe the generation of a replication-defective adenoviral vector, AdRSVIDS, which is able to express in vitro high levels of iduronate-2-sulfatase. After infection, accumulation of mucopolysaccharides in treated Hunter cells was normalized. Furthermore, endocytosis of the transduced IDS did occur via the mannose-6-phosphate (M6P) receptor. Since no animal model for the disease is available, we developed a system based on the generation of derma-equivalent which enables us to verify the expression of high levels of sulfatase up to 30 days after infection.
RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 21 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1996:391236 CAPLUS
DN 125:80131

TI Lysosomal targeting of palmitoyl-protein thioesterase
AU Verkruyse, Linda A.; Hofmann, Sandra L.
CS Department Internal Medicine, University Texas Southwestern Medical Center, Dallas, TX, 75235-8593, USA
SO Journal of Biological Chemistry (1996), 271(26), 15831-15836
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
AB Palmitoyl-protein thioesterase is a newly described long chain fatty-acid hydrolase that removes fatty acyl groups from modified cysteines in proteins. We have recently identified palmitoyl-protein thioesterase as the defective enzyme in the recessive hereditary neurol. degenerative disorder infantile neuronal ceroid lipofuscinosis (Vesa, J., Hellsten, E., Verkruyse, L. A., Camp, L. A., Rapola, J., Santavuori, P., Hofmann, S. L., and Peltonen, L. (1995) Nature 376, 584-587). A defect in a lysosomal enzyme had been postulated for the disease, but until recently, the relevant defective lysosomal enzyme had not been identified. In this paper, we present evidence for the lysosomal localization of palmitoyl-protein thioesterase. We show that COS cells take up exogenously supplied palmitoyl-protein thioesterase intracellularly and that the cellular uptake is blocked by mannose 6-phosphate, a hallmark of lysosomal enzyme trafficking. The enzyme contains endoglycosidase H-sensitive oligosaccharides that contain phosphate groups. Furthermore, palmitoyl-protein thioesterase cosediments with lysosomal enzyme markers by Percoll d. gradient centrifugation. Interestingly, the pH optimum for the enzyme is in the neutral range, a property shared by two other lysosomal enzymes that remove post-translational protein modifications. These findings suggest that palmitoyl-protein thioesterase is a lysosomal enzyme and that infantile neuronal ceroid lipofuscinosis is properly classified as a lysosomal storage disorder.

L20 ANSWER 22 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1995:818365 CAPLUS
DN 123:225192
TI Abnormal synthesis of dolichol-linked oligosaccharides in carbohydrate-deficient glycoprotein syndrome
AU Krasnewich, Donna M.; Holt, Gordon D.; Brantly, Mark; Skovby, Flemming; Redwine, Jeff; Gahl, William A.
CS Section Human Biochemical Genetics, Human Genetics Branch, NICHD, National Institutes Health, Bedford, MA, USA
SO Glycobiology (1995), 5(5), 503-10
CODEN: GLYCE3; ISSN: 0959-6658
PB Oxford University Press
DT Journal
LA English
AB Carbohydrate-deficient glycoprotein syndrome (CDGS) is a rare metabolic disorder presenting in infancy with severe neurol. involvement and variable multisystemic abnormalities. Diagnosis relies upon the detection of abnormal serum glycoprotein isoforms on isoelec. focusing (IEF) gels. Carbohydrate structural analyses were performed on the N-linked oligosaccharides on serum α1-antitrypsin (α-1AT) from two Danish children with classical type I CDGS. Following preparative gel electrophoresis of α-1AT isoforms, oligosaccharide change and monosaccharide composition anal. revealed increased glycosylation heterogeneity in CDGS compared with normal α-1AT. CDGS α-1AT isoforms bore N-glycans comigrating with monosialylated stds., while normal α-1AT oligosaccharides comigrated with both mono-and disialylated stds. While the monosaccharide contents of normal α-1AT isoforms were relatively uniform, those of CDGS α-1AT isoforms varied widely, and many were relatively mannose enriched. The mannose-rich oligosaccharides of CDGS α-1AT were not typical oligomannose structures since they were not released by

endo-β-N-acetylglucosaminidase H (endo H) digestion. Metabolic labeling of CDGS fibroblasts with [3H]mannose showed lower than normal intracellular total mannose, free mannose and phosphorylated mannose species, as well as diminished [3H] mannose incorporation into dolichol-linked and protein-linked oligosaccharides. In addition, the glycans liberated from CDGS dolichol-linked oligosaccharides were significantly truncated compared with those from normal fibroblasts. These data suggest that our type I CDGS patients produce abnormal N-linked oligosaccharides due to impaired biosynthesis of dolichol-oligosaccharide precursors.

L20 ANSWER 23 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1991:227367 CAPLUS
DN 114:227367
TI Immunoassay, lectin, and kit for the diagnosis of neurological demyelination disorders, especially multiple sclerosis
IN Zanetta, Jean Pierre; Warter, Jean Marie; Kuchler, Sabine; Vincendon, Guy
PA Centre National de la Recherche Scientifique, Fr.
SO Eur. Pat. Appl., 11 pp.
CODEN: EPXXDW
DT Patent
LA French
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 416984	A1	19910313	EP 1990-402399	19900830
	EP 416984	B1	19930616		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE				
	FR 2651581	A1	19910308	FR 1989-11667	19890906
	WO 9103737	A1	19910321	WO 1990-FR638	19900830
	W: AU, FI, JP, NO				
	AU 9064214	A	19910408	AU 1990-64214	19900830
	AU 648516	B2	19940428		
	JP 05500858	T	19930218	JP 1990-513133	19900830
	JP 3032290	B2	20000410		
	AT 90791	T	19930715	AT 1990-402399	19900830
	ES 2057476	T3	19941016	ES 1990-402399	19900830
	US 5225352	A	19930706	US 1990-577884	19900905
	CA 2024731	A1	19910307	CA 1990-2024731	19900906
	NO 9200852	A	19920304	NO 1992-852	19920304
PRAI	FR 1989-11667	A	19890906		
	EP 1990-402399	A	19900830		
	WO 1990-FR638	A	19900830		

AB Neurol. demyelination disorders, especially multiple sclerosis (MS), are diagnosed by (1) contacting patient fluid (cerebrospinal fluid or blood) with an endogenous lectin having an affinity for glycans rich in mannose or their subunit glycoproteins, especially cerebellar soluble lectin (CSL) or its protein component;
and (2) detecting formed immunol. complex. A kit for diagnosing MS and neurol. demyelination disorders comprises a CSL-type lectin or subunit and solvents and agents necessary for the assay. Nitrocellulose filters containing transferred partially purified CSL from PAGE were incubated with cerebrospinal fluid samples and then labeled with goat anti-human IgG labeled with horseradish peroxidase or alkaline phosphatase. Colored bands were developed in 47/51 samples of patients clin. diagnosed with MS. The test had a specificity of 85% and a sensitivity of 93.5%.

L20 ANSWER 24 OF 24 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1961:13849 CAPLUS
DN 55:13849
OREF 55:2781g-i,2782a
TI Determination of neuraminic (sialic) acid, glucose, and fructose in spinal fluid

AU Papadopoulos, Nicholas M.; Hess, Walter C.
CS Georgetown Univ., Washington, DC
SO Archives of Biochemistry and Biophysics (1960), 88, 167-71
CODEN: ABBIA4; ISSN: 0003-9861
DT Journal
LA Unavailable
AB cf. CA 53, 9419a. The sialic acids (I) consisting of N-acetylneuraminic acid (II) and related compds. are estimated in spinal fluid by the color produced with Bial's reagent in a spectrophotometer. Fructose (III) also reacts, so III is estimated by its color reaction with resorcinol in HCl read spectrophotometrically. Glucose (IV) interferes to a lesser extent and is estimated by a peroxidase oxidation in presence of o-dianisidine. Curves indicate that the color reaction with the orcinol reagent decreases in the order II, III, and IV. By correcting for the III and IV, accurate values for I (as II) are determined The optical d. is at a maximum at 570 mμ in all
3
cases. Free ribose and deoxyribose also give colors but are not found in spinal fluid. Galactose, fucose, mannose, glucosamine, inositol, and pyruvic and lactic acids gave no color in the concns. occurring in spinal fluid. II gave no color in the resorcinol test for III, but large amts. of IV might produce some color. The average of 10 detns. on normal spinal fluids gave values of IV 57, III 3.4, and I 1.8 mg. %. The range for I was 1.4-2.25 mg. %. In 10 cases of neurological disorders, the average values for free and bound (esterified) I in spinal fluid were 1.12 and 0.70 mg. %, resp. II was demonstrated (after hydrolysis) in these samples by paper chromatography. An ion-exchange method (Svennerholm, CA 53, 15172h) gave average I values about 12% less than the new method. 15 references.

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=> s l1 and gulose
            642 GULOSE
              4 GULOSES
            642 GULOSE
                  (GULOSE OR GULOSES)
L21           2 L1 AND GULOSE

=> dis k21 1-2 bib abs
'K21' IS NOT A VALID FORMAT FOR FILE 'CAPLUS'

The following are valid formats:

ABS --------- GI and AB
ALL --------- BIB, AB, IND, RE
APPS -------- AI, PRAI
BIB --------- AN, plus Bibliographic Data and PI table (default)
CAN --------- List of CA abstract numbers without answer numbers
CBIB -------- AN, plus Compressed Bibliographic Data
CLASS ------- IPC, NCL, ECLA, FTERM
DALL -------- ALL, delimited (end of each field identified)
DMAX -------- MAX, delimited for post-processing
FAM --------- AN, PI and PRAI in table, plus Patent Family data
FBIB -------- AN, BIB, plus Patent FAM
IND --------- Indexing data
IPC --------- International Patent Classifications
MAX --------- ALL, plus Patent FAM, RE
PATS -------- PI, SO
SAM --------- CC, SX, TI, ST, IT
SCAN -------- CC, SX, TI, ST, IT (random display, no answer numbers;
              SCAN must be entered on the same line as the DISPLAY,
              e.g., D SCAN or DISPLAY SCAN)
STD --------- BIB, CLASS

IABS -------- ABS, indented with text labels
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IALL -------- ALL, indented with text labels
IBIB -------- BIB, indented with text labels
IMAX -------- MAX, indented with text labels
ISTD -------- STD, indented with text labels

OBIB -------- AN, plus Bibliographic Data (original)
OIBIB ------- OBIB, indented with text labels

SBIB -------- BIB, no citations
SIBIB ------- IBIB, no citations

HIT --------- Fields containing hit terms
HITIND ------ IC, ICA, ICI, NCL, CC and index field (ST and IT)
              containing hit terms
HITRN ------- HIT RN and its text modification
HITSTR ------ HIT RN, its text modification, its CA index name, and
              its structure diagram
HITSEQ ------ HIT RN, its text modification, its CA index name, its
              structure diagram, plus NTE and SEQ fields
FHITSTR ----- First HIT RN, its text modification, its CA index name, and
              its structure diagram
FHITSEQ ----- First HIT RN, its text modification, its CA index name, its
              structure diagram, plus NTE and SEQ fields
KWIC -------- Hit term plus 20 words on either side
OCC --------- Number of occurrence of hit term and field in which it occurs

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codes.  For a list of the display field codes, enter HELP DFIELDS at
an arrow prompt (=>).  Examples of formats include: TI; TI,AU; BIB,ST;
TI,IND; TI,SO.  You may specify the format fields in any order and the
information will be displayed in the same order as the format
specification.

All of the formats (except for SAM, SCAN, HIT, HITIND, HITRN, HITSTR,
FHITSTR, HITSEQ, FHITSEQ, KWIC, and OCC) may be used with DISPLAY ACC
to view a specified Accession Number.
ENTER DISPLAY FORMAT (BIB):end

=> dis l21 1-2 bib abs

L21  ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2007 ACS on STN
AN   2004:290472  CAPLUS
DN   140:264527
TI   Methods and compositions for treatment of neurological
     disorder
IN   Benowitz, Larry I.
PA   Children's Medical Center Corporation, USA
SO   PCT Int. Appl., 59 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.          DATE
     --------------      ----   --------    --------------------     --------
PI   WO 2004028468        A2    20040408    WO 2003-US30466          20030925
     WO 2004028468        A3    20041021
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM,
             PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
             TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES,
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FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,
BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

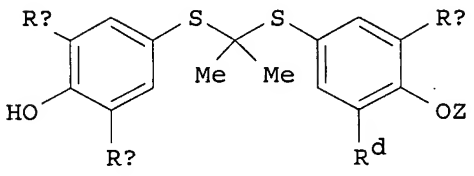
	CA 2499170	A1	20040408	CA 2003-2499170	20030925
	AU 2003272728	A1	20040419	AU 2003-272728	20030925
	EP 1542702	A2	20050622	EP 2003-754929	20030925
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	CN 1703227	A	20051130	CN 2003-825428	20030925
	JP 2006503847	T	20060202	JP 2004-540004	20030925
	US 2005256059	A1	20051117	US 2005-528685	20050718
PRAI	US 2002-414063P	P	20020927		
	WO 2003-US30466	W	20030925		

AB The invention provides methods and compns. for producing a neurosalutary effect in a subject useful for the treatment of neurol. disorders, including retinal and optic nerve damage, in a subject in need thereof. The method includes administration to a subject a therapeutically effective amount of a hexose, such as mannose.

L21 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:713364 CAPLUS
DN 135:267271
TI Probucol-related thioketals and thioethers for inhibiting the expression of VCAM-1, preparation, and therapeutic use
IN Meng, Charles Q.; Hoong, Lee K.; Somers, Patricia K.
PA Atherogenics, Inc., USA
SO PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 4

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001070757	A2	20010927	WO 2001-US9049	20010321
	WO 2001070757	A3	20020314		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2403823	A1	20010927	CA 2001-2403823	20010321
	EP 1289944	A2	20030312	EP 2001-920617	20010321
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	JP 2003528109	T	20030924	JP 2001-568958	20010321
	AU 2001247651	B2	20070104	AU 2001-247651	20010321
	AU 2002300328	A1	20021219	AU 2002-300328	20020730
PRAI	US 2000-191046P	P	20000321		
	AU 1998-74851	A3	19980514		
	WO 2001-US9049	W	20010321		

OS MARPAT 135:267271
GI



I

AB Probucol-related thioketals and thioethers are provided that inhibit the expression of VCAM-1, and which can be used in the treatment of VCAM-1-mediated diseases, including inflammatory disorders, cardiovascular diseases, ocular diseases, autoimmune diseases, neurol. disorders, and cancer. Compds. of the invention include I [Ra-Rd = H, (un)substituted alkyl, (un)substituted aryl, etc.; Z = (un)substituted carbohydrate, (un)substituted alditol, (un)substituted C1-10 alkyl terminated by sulfonic acid, etc.]. The compds. also can be used to treat hyperlipidemia and/or hypercholesterolemia. Compound preparation is described.

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=> s l1 and glucose-6-phosphate
        434139 GLUCOSE
           835 GLUCOSES
        434318 GLUCOSE
                  (GLUCOSE OR GLUCOSES)
       3981687 6
        582010 PHOSPHATE
        129741 PHOSPHATES
        632078 PHOSPHATE
                  (PHOSPHATE OR PHOSPHATES)
         28766 GLUCOSE-6-PHOSPHATE
                  (GLUCOSE(W)6(W)PHOSPHATE)
L22          16 L1 AND GLUCOSE-6-PHOSPHATE

=> dis l22 1-16 bib abs

L22  ANSWER 1 OF 16  CAPLUS  COPYRIGHT 2007 ACS on STN
AN   2006:524606  CAPLUS
DN   145:117320
TI   Oxidative stress-mediated macromolecular damage and dwindle in antioxidant
     status in aged rat brain regions: Role of L-carnitine and -α-lipoic
     acid
AU   Muthuswamy, Anusuya Devi; Vedagiri, Kokilavani; Ganesan, Murali;
     Chinnakannu, Panneerselvam
CS   Taramani Campus, Department of Medical Biochemistry, Dr. AL. Mudaliar Post
     Graduate Institute of Basic Medical Sciences, University of Madras,
     Chennai, 600 113, India
SO   Clinica Chimica Acta (2006), 368(1-2), 84-92
     CODEN: CCATAR; ISSN: 0009-8981
PB   Elsevier Ltd.
DT   Journal
LA   English
AB   Background: The free radical theory of aging has significant relevance in
     a number of age-related neurol. disorders. Too many free
     radicals create cellular pollution that shuts down energy levels. They
     have also been implicated in the loss of physiol. functioning associated with
     the aging of post mitotic cells such as the brain. The activities of
     enzymic antioxidative defenses decrease in rat brain may be possible
     causes of age-associated increase in oxidative damage to macromols. Methods:
     We determined whether DL-α-lipoic acid (100 mg/kg body weight/day), and
     -carnitine (300 mg/kg body weight/day) together when administered for 30 days
     declines the rate of oxidative stress-mediated macromol. damages such as
     lipid peroxidn. (LPO), protein carbonyl (PCO) and DNA protein cross-links
     in different anat. regions (cortex, striatum and hippocampus). The
     activities of antioxidant enzymes in programmed aging were evaluated in
     the cortex, striatum and hippocampus of young and aged rat brain regions.
     Results: Aged rats elicited a significant decline in the antioxidant
     status and increase in LPO, PCO and DNA protein cross-links as compared to
     young rats in all the 3 brain regions. The increase in LPO, PCO and DNA
     protein cross-links were (35.8%, 35.6%, 43.5%) in cortex, (32.5%, 40.3%,
     29.8%) in striatum and (62.7%, 42.4%, 34.9%) in hippocampus, resp., in
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aged rats as compared to young rats. Co-supplementation of carnitine and lipoic acid was found to be effective in reducing brain regional LPO, PCO and DNA protein cross-links and in increasing the activities of enzymic antioxidants in aged rats to near normalcy. Conclusion: The combination of -carnitine and lipoic acid overcame the oxidative stress induced rate of lipid peroxidn., protein carbonyl formation, accumulation of DNA protein cross-links and deficits in antioxidant enzyme activities in various brain regions of aged rats.

RE.CNT 60 THERE ARE 60 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 2 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:290472 CAPLUS
DN 140:264527
TI Methods and compositions for treatment of neurological disorder
IN Benowitz, Larry I.
PA Children's Medical Center Corporation, USA
SO PCT Int. Appl., 59 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004028468	A2	20040408	WO 2003-US30466	20030925
	WO 2004028468	A3	20041021		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	CA 2499170	A1	20040408	CA 2003-2499170	20030925
	AU 2003272728	A1	20040419	AU 2003-272728	20030925
	EP 1542702	A2	20050622	EP 2003-754929	20030925
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	CN 1703227	A	20051130	CN 2003-825428	20030925
	JP 2006503847	T	20060202	JP 2004-540004	20030925
	US 2005256059	A1	20051117	US 2005-528685	20050718
PRAI	US 2002-414063P	P	20020927		
	WO 2003-US30466	W	20030925		

AB The invention provides methods and compns. for producing a neurosalutary effect in a subject useful for the treatment of neurol. disorders, including retinal and optic nerve damage, in a subject in need thereof. The method includes administration to a subject a therapeutically effective amount of a hexose, such as mannose.

L22 ANSWER 3 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2001:101125 CAPLUS
DN 134:157573
TI Dithiolthione compounds for the treatment of neurological disorders and for memory enhancement
IN Prendergast, Patrick T.; Armstrong, Paul
PA Ire.
SO PCT Int. Appl., 109 pp.
CODEN: PIXXD2
DT Patent
LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001009118	A2	20010208	WO 2000-IB1146	20000728
	WO 2001009118	A3	20011122		
	W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	AU 2000064625	A	20010219	AU 2000-64625	20000728
	US 2004053989	A1	20040318	US 2003-612476	20030702
PRAI	US 1999-145964P	P	19990729		
	IE 2000-302	A	20000413		
	IE 2000-304	A	20000413		
	US 2000-198338P	P	20000418		
	US 2000-627641	B1	20000728		
	WO 2000-IB1146	W	20000728		

OS MARPAT 134:157573

AB The invention provides methods to treat neurol. disorders such as Alzheimer's disease, or to slow the progression of such diseases, or to treat and/or prevent other disorders as disclosed in the specification, by administering to patients, or delivering to the tissues of such patients, oltipraz or related 1,2-dithiole-3-thiones. The effects of oltipraz on Aβ1-42 neurotoxicity, oxidative stress, removal of iron from tissues, localization of 8-hydroxyguanosine (predominantly derived from •OH attack of guanidine), mitochondrial damage as well as its antiprotozoal activity were examined Synthesis of oltipraz is presented.

L22 ANSWER 4 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN

AN 1998:725501 CAPLUS

DN 130:192487

TI Molecular basis of neurological dysfunction coupled with hemolytic anemia in human glucose-6-phosphate isomerase (GPI) deficiency

AU Kugler, Wilfried; Breme, Kathrin; Laspe, Petra; Muirhead, Hilary; Davies, Christopher; Winkler, Heinz; Schroter, Werner; Lakomek, M.

CS Universitats-Kinderklinik, Robert-Koch-Strasse 40, Gottingen, D-37075, Germany

SO Human Genetics (1998), 103(4), 450-454
CODEN: HUGEDQ; ISSN: 0340-6717

PB Springer-Verlag

DT Journal

LA English

AB Glucose-6-phosphate isomerase (GPI) deficiency, an autosomal recessive genetic disorder with the typical manifestation of nonspherocytic hemolytic anemia, can be associated in some cases with neurol. impairment. GPI was found to be identical to neuroleukin (NLK), which has neurotrophic and lymphokine properties. To focus on the effects of GPI mutations on the central nervous system through an effect on neuroleukin activity, the authors analyzed DNA isolated from 2 patients with severe GPI deficiency, 1 of them with addnl. neurol. deficits, and their families. The neurol. affected patient (GPI Homburg) is compound heterozygous for a 59 A→C (H20P) and a 1016 T→C (L339P) exchange. Owing to the insertion of Pro, the H20P and L339P mutations are likely to affect the folding and activity of the enzyme. In the 2nd family studied, the 2 affected siblings showed no neurol. symptoms. The identified mutations are 1166 A→G (H389R) and 1549 C→G (L517V), which are located at the subunit interface. The authors propose that mutations

that lead to incorrect folding destroy both catalytic (GPI) and neurotrophic (NLK) activities, thereby leading to the observed clin. symptoms (GPI Homburg). Those alterations at the active site, however, that allow correct folding retain the neurotrophic properties of the mol. (GPI Calden).

RE.CNT 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 5 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1998:682105 CAPLUS
DN 129:298408
TI Nitrosylation to inactivate apoptotic enzymes, and therapeutic caspase-like peptide
IN Lipton, Stuart A.; Troy, Carol M.
PA The Children's Medical Center Corp., USA
SO PCT Int. Appl., 20 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9843621	A1	19981008	WO 1998-US6287	19980331
	W: CA, JP				
	RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	EP 979073	A1	20000216	EP 1998-913316	19980331
	R: DE, ES, FR, GB, IT				
	JP 2001518096	T	20011009	JP 1998-541915	19980331
	US 2002106404	A1	20020808	US 2002-55417	20020122
	US 2004265369	A1	20041230	US 2004-839434	20040505
	US 2007218121	A1	20070920	US 2006-594565	20061108
PRAI	US 1997-42144P	P	19970331		
	US 1998-52826	B1	19980331		
	WO 1998-US6287	W	19980331		
	US 2002-55417	A1	20020122		
	US 2004-839434	B1	20040505		

OS MARPAT 129:298408
AB S-nitrosylation (reaction of nitric oxide [NO] species with critical cysteine sulfhydryl groups of a caspase [RS] to form RS-NO) inhibits caspase activity and thereby ameliorates apoptosis not only in neuronal cells, but also in other tissues. Addnl., ICE-like (caspase-like) sequence ICARG is used to protect from excitotoxic neuronal damage and neurol. as well as non-neurol. and non-ophthalmol. indications characterized by undesired apoptosis.

RE.CNT 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 6 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1998:450407 CAPLUS
DN 129:160675
TI Production of D-[1-13C]glucose from [13C]formaldehyde and D-ribose-5-phosphate using an enzymic system
AU Maeda, Hidekatsu; Takata, Kohhei; Toyoda, Atsushi; Shibata, Kunihiko
CS Department of Bioengineering, Faculty of Engineering, Soka University, Tokyo, 192-8577, Japan
SO Journal of Fermentation and Bioengineering (1998), 85(5), 536-538
CODEN: JFBIEX; ISSN: 0922-338X
PB Society for Fermentation and Bioengineering, Japan
DT Journal
LA English
AB D-[1-13C]Glucose is useful for studies on tracing by means of 13C-magnetic resonance imaging (MRI) the fate of a metabolite in the brain, and this technique is expected to become a sophisticated clin. tool for the diagnosis of neurol. disorders. For this purpose, a

more efficient method of producing inexpensive 13C-labeled D-glucose is necessary, and was investigated. When a mixture of the labeled D-fructose-6-phosphate (F-6-P) and D-glucose-6-phosphate (G-6-P) prepared from D-ribose-5-phosphate and [13C]formaldehyde is dephosphorylated by potato acid phosphatase, a combination of potato acid phosphatase, phosphoglucose isomerase, and BaCl2 was found to be effective in improving the yield of D-glucose and shortening the reaction time. The maximum yield of labeled D-glucose from the mixture of labeled F-6-P and G-6-P was 84%. Two peaks indicative of labeled D-glucose with 13C incorporated at the C-1 position were confirmed by the NMR spectroscopy.

RE.CNT 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 7 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1997:663597 CAPLUS
DN 127:344475
TI Erythrocyte pyruvate kinase- and glucose phosphate isomerase deficiency: perturbation of glycolysis by structural defects and functional alterations of defective enzymes and its relation to the clinical severity of chronic hemolytic anemia
AU Lakomek, Max; Winkler, Heinz
CS Universitaets-Kinderklinik und Poliklinik and Max Planck Institut fuer biophysikalische Chemie, Gottingen, Germany
SO Biophysical Chemistry (1997), 66(2-3), 269-284
CODEN: BICIAZ; ISSN: 0301-4622
PB Elsevier
DT Journal; General Review
LA English
AB A review, with 54 refs., of primarily the authors' work. The pathogenesis of two metabolic disorders caused by enzyme defects in the red blood cell leading to hemolytic anemia, and in some cases of glucose phosphate isomerase (GPI) deficiency addnl. to neurol. impairment was investigated. Rheol. studies were performed to determine the influence of a shortage of energy on the deformability of the erythrocytes. The functions of the enzymes were determined by studying the enzyme kinetics, the temperature dependence of the enzyme activity, and the migration of the proteins in an elec. field. A detailed mol. genetic anal. of the gene encoding for the given protein allowed the detection of mutations involving amino acid exchanges which cause alterations of the protein structure. For both enzyme deficiencies, a good correlation was found between the structural changes (usually caused by single point mutations in the gene), the altered function of the enzymes and the severity of the clin. picture. The exchange of amino acids close to either the active site or the regulatory domain results in a decreased turnover as well as an alteration of the regulatory properties of the enzymes; this usually leads to an increased severity of the disease. Increased concns. of glucose 6-phosphate (G-6-P), found in all red blood cells of patients suffering from hemolytic anemia caused by pyruvate kinase (PK) and GPI deficiency, correlate well with the severity of the clin. picture, apparently reflecting the degree of the perturbation of glycolysis. This results in a lack of the energy donor ATP; this leads then to a destabilization of the red cell membrane which causes earlier lysis of the red blood cell, which in turn gives rise to hemolytic anemia of variable degrees. One patient with neurol. symptoms has been studied so far biochem. and at the mol. genetic level. The point mutations found in this patient's GPI gene support the idea that GPI may have a neurol. function in addition to its role in the carbohydrate metabolism; this is due to the presence of a monomeric sequence analog called neuroleukin (NLK). The mutations apparently lead to the incorrect folding of this neurotrophic factor, and thus destroy the neurol. activity.

RE.CNT 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L22 ANSWER 8 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1997:338610 CAPLUS
DN 127:16017
TI Glutathione in the brain: disorders of glutathione metabolism
AU Cooper, Arthur J. L.
CS Cornell University Medical College, New York, NY, USA
SO Molecular and Genetic Basis of Neurological Disease (2nd Edition) (1997), 1195-1230. Editor(s): Rosenberg, Roger N. Publisher: Butterworth-Heinemann, Boston, Mass.
CODEN: 64KBAL
DT Conference; General Review
LA English
AB A review, with 478 refs. Glutathione (GSH) occurs in all regions of the brain at levels in the range of 1-3 mM; low levels (25 μM) occur in cerebrospinal fluid. All the enzymes of the γ-glutamyl cycle, glutathione disulfide (GSSG) reductase, GSH peroxidases, thiol transferases, and GSH S-transferases (GSTs), are present in brain. Compared with many other tissues, brain has somewhat limited mechanisms for protection against the effects of reactive oxygen compds. and free radicals. GSH metabolism in brain is probably compartmentalized. Astrocytes contain substantial levels of GSH and GSTs. The available information suggests that astrocytes function importantly in protecting brain against reactive oxygen species and other toxic compds. Recent evidence suggests that nerve endings also contain GSH and that GSH is released from this pool. Astrocytes contain high-affinity binding sites for GSH. Moreover, GSH appears to be an endogenous agonist of the N-methyl-D-aspartate (NMDA) glutamate receptor, and GSH may be a neurotransmitter in the spinal cord. Small quantities of GSH may be transported intact across the blood-brain barrier (BBB) in adult animals, but the contribution of this process to whole brain GSH is probably small. Total brain GSH has been reported to turn over with a half-life of 70 h. A portion of the brain GSH, however, turns over rapidly (half-life of approx. 30 min). This rapidly turned over GSH pool may be in the astrocytes. Depletion of brain GSH has been accomplished by administration of L-buthionine-S,R-sulfoximine (BSO, a selective inhibitor of γ-glutamylcysteine synthetase) or of compds. that react with GSH in the GST-catalyzed reaction. Methods for increasing brain GSH levels have also been devised. γ-Glutamyl transpeptidase is present in high levels in brain capillaries and associated pericytes, and this enzyme may play a role in the normal functioning of the intact BBB. Several inborn errors of the γ-glutamyl cycle are known, including defects of glutathione synthetase, γ-glutamylcysteine synthetase, γ-glutamyl transpeptidase, and 5-oxoprolinase. Other conditions that affect GSH metabolism, such as GSH peroxidase deficiency, GSSG reductase deficiency, and glucose-6-phosphate dehydrogenase deficiency, are discussed here. Many of the patients with such defects have neurol. abnormalities, which attests to the importance of GSH in normal brain function.

L22 ANSWER 9 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1996:438559 CAPLUS
DN 125:131350
TI Drug-induced methemoglobinemia: Treatment issues
AU Coleman, Michael D.; Coleman, Nicholas A.
CS Department Pharmaceutical Sciences, Aston University, Birmingham, UK
SO Drug Safety (1996), 14(6), 394-405
CODEN: DRSAEA; ISSN: 0114-5916
PB Adis
DT Journal; General Review
LA English
AB A review with 84 refs. In normal erythrocytes, small quantities of metHb are formed constantly and are continuously reduced, almost entirely by the reduced nicotine adenine dinucleotide (NADH) diaphorase system, rather than the reduced nicotine adenine dinucleotide phosphate (NADPH)

diaphorase system. Methemoglobinemias are usually the result of xenobiotics, either those that may directly oxidize Hb or those that require metabolic activation to an oxidizing species. The most clin. relevant direct metHb formers include local anesthetics (such as benzocaine and, to a much lesser extent, prilocaine) as well as amyl nitrite and iso-Bu nitrite, which have become drugs of abuse. Indirect, or metabolically activated, metHb formation by dapsone and primaquine may cause adverse reactions. The clin. consequences of methemoglobinemia are related to the blood level of metHb; dyspnea, nausea and tachycardia occur at metHb levels of ≥30%, while lethargy, stupor and deteriorating consciousness occur as metHb levels approach 55%. Higher levels may cause cardiac arrhythmias, circulatory failure and neurol. depression, while levels of 70% are usually fatal. Cyanosis accompanied by a lack of responsiveness to 100% oxygen indicates a diagnosis of methemoglobinemia, which should be confirmed using a CO-oximeter. Pulse oximeters do not detect metHb and may give a misleading impression of patient oxygenation. Methemoglobinemia is treated with i.v. methylene blue (methylthioninium chloride; 1 to 2 mg/kg of a 1% solution). If the patient does not respond, perhaps because of glucose-6-phosphate dehydrogenase (G6PD) deficiency or continued presence of toxin, admission to an intensive care unit and exchange transfusion may be required. Dapsone-mediated chronic metHb formation can be reduced by co-administration of cimetidine to aid patient tolerance. Increasing knowledge and awareness of drug-mediated acute methemo-globinemia among physicians should lead to prompt diagnosis and treatment of this potentially life-threatening condition.

L22 ANSWER 10 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1994:677655 CAPLUS
DN 121:277655
TI Iron and aluminum homeostasis in neural disorders
AU Joshi, Jayant G.; Dhar, Madhu; Clauberg, Martin; Chauthaiwale, Vijay
CS Department Biochemistry, University Tennessee, Knoxville, TN, 37996-0840, USA
SO Environmental Health Perspectives Supplements (1994), 102(SUPPL. 3), 207-13
CODEN: EHPSEO; ISSN: 1078-0475
DT Journal; General Review
LA English
AB A review with 53 refs. The brain is the most compartmentalized organ. It is also highly aerobic. Because nerve cells grow but do not regenerate, the brain is the organ best suited for the accumulation of metabolic errors colocalized in specific areas of the brain over an extended period. Alzheimer's disease (AD) is primarily a neurol. disorder of the elderly. It is suggested that this disorder results from the accumulation of such errors, and that AD onset aluminum and iron contribute to but do not necessarily initiate the onset of the disease. In vitro and in vivo evidence summarized here suggests that this is effected by interfering in the utilization of glucose and glucose -6-phosphate, and sequestration of iron by ferritin. β-Amyloid precursor proteins (β-APPs) are normal components of the human brain and some other tissues. Proteolysis of these, presumably by serine proteases, generates a 39 to 42 amino acid long peptide, the α-amyloid (β-AP). In AD brains, β-AP aggregates into plaque, the hallmark of AD brains. Some of the α-APPs also contain a 56 amino acid long segment which inhibits serine proteases. The authors show that in vitro, at pH 6.5, aluminum activates β-chymotrypsin 2-fold and makes it dramatically resistant to protease inhibitors such as bovine pancreatic trypsin inhibitor (bPTI) or its mimic present in the β-amyloid precursor proteins (β-APPs). Iron and oxygen are reported to favor crosslinking of β-AP in vitro. Because iron and ferritin are components of neurotic plaques, and acidic pH are reported in AD brains, the authors suggest that deregulation of iron and aluminum homeostasis permit their colocalization, and contribute to the

accumulation of metabolic errors leading to neuronal disorders including the formation of AD (senile) plaques.

L22 ANSWER 11 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1994:47164 CAPLUS
DN 120:47164
TI Transcriptional organization of a 450-kb region of the human X chromosome in Xq28
AU Bione, S.; Tamanini, F.; Maestrini, E.; Tribioli, C.; Poustka, A.; Torri, G.; Rivella, S.; Toniolo, D.
CS Consigl. Naz. Ric., Ist. Genet. Biochim. Evol., Pavia, 27100, Italy
SO Proceedings of the National Academy of Sciences of the United States of America (1993), 90(23), 10977-81
CODEN: PNASA6; ISSN: 0027-8424
DT Journal
LA English
AB The transcriptional organization of a 450-kb gene cluster in human chromosome Xq28, flanked by the glucose-6-phosphate dehydrogenase and the color vision genes, was studied. CpG islands previously identified and mapped to distal Xq28 have helped in construction of a continuous contig of cosmids and in identification of cDNAs corresponding to 8 transcripts. Thirteen to 16 small genes with CpG islands are clustered in a region of 250-300 kb. Many are highly expressed in muscle or brain and may be the genes responsible for muscle or neurol. disorders mapped to distal Xq28. In this region of the genome, genes not related in sequence are organized in transcriptional domains of 100 kb and this organization may be important for establishing and regulating gene expression in relation to tissue distribution and X chromosome inactivation.

L22 ANSWER 12 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1983:610312 CAPLUS
DN 99:210312
TI The rate of utilization of glucose via hexose monophosphate shunt in brain
AU Gaitonde, M. K.; Evison, E.; Evans, G. M.
CS Med. Sch., St. George's Hosp., London, UK
SO Journal of Neurochemistry (1983), 41(5), 1253-60
CODEN: JONRA9; ISSN: 0022-3042
DT Journal
LA English
AB The concentration of 6-phosphogluconate in the brain increased from 0-24 nmol/g in the controls to 1430 and 1506 nmol/g in rats treated with 50 mg 6-aminonicotinamide (I)/kg body weight A dose-dependent increase in the concns. of glucose and glucose 6-phosphate as well as of 6-phosphogluconate was found in the brains of I-treated rats. The biochem. changes and symptoms of neurol. disorder in I-treated rats were not due to hypothermia. The rate of utilization of glucose via the hexose monophosphate shunt was determined by isolation of gluconate from 6-phosphogluconate and measurement of its 14C content at short time intervals after injection of [U-14C]glucose into I-treated rats; it was 16.5 nmol glucose utilized/min/g brain, and represented .apprx.2.3% of the overall utilization of glucose in the brain. A highly significant correlation was observed between the concentration of 6-phosphogluconate and the concentration of glucose 6-phosphate and free glucose. The validity of this correlation was supported by the results of previous investigations involving several other treatments.

L22 ANSWER 13 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1971:527946 CAPLUS
DN 75:127946
OREF 75:20191a
TI Histopathologic and enzyme histochemical observations of the

cuprizone-induced brain edema
AU Kesterson, James W.; Carlton, William W.
CS Sch. Vet. Sci. Med., Purdue Univ., Lafayette, IN, USA
SO Experimental and Molecular Pathology (1971), 15(1), 82-96
CODEN: EXMPA6; ISSN: 0014-4800
DT Journal
LA English
GI For diagram(s), see printed CA Issue.
AB Progressively severe and disseminated status spongiosus and astrocytosis occurred in the brain of mice fed the neurotoxin cuprizone (I) (0.3% of the diet). Normal astrocytes showed little or no oxidative enzyme activity, whereas pathol. astrocytes exhibited strong activity for the various enzymes studied. Weak glutamate dehydrogenase (GDH) activity was found in astrocytes after only 3 days of I feeding, followed (after ≥5 days) by strong activity for GDH, NAD diaphorase, and lactic dehydrogenase. Increased activity of NADP diaphorase, glucose-6-phosphate dehydrogenase, and succinic dehydrogenase was observed in pathol. astrocytes late in the experiment, but these enzymes never reached the level of GDH and NAD diaphorase. The edema and the increased oxidative enzymes of the astrocytes generally paralleled each other in severity.

L22 ANSWER 14 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1968:67321 CAPLUS
DN 68:67321
OREF 68:12971a
TI Oxidative metabolism of the brain in experimental cerebral edema
AU Pausescu, Exacustodian; Schwartz, Beno; Chirvasie, Rodica; Dinca, Alice
CS Clin. Hosp. Fundeni, Bucharest, Rom.
SO Experimental Neurology (1967), 19(4), 455-62
CODEN: EXNEAC; ISSN: 0014-4886
DT Journal
LA English
AB In dogs of both sexes cerebral edema was induced by the intraarterial injection of grains of polyurethan. The animals were killed after 1 or 2 days when in most of the animals neurological disorders had developed. Succinic dehydrogenase (I), NAD-specific isocitric dehydrogenase (II), glutamyl transferase (III), glucose-6-phosphate dehydrogenase (IV), α-ketoglutaric dehydrogenase (V), and phosphatase activities were studied in gray and white matter samples taken from both brain hemispheres. The ability of the cerebral tissue to oxidize in vitro isocitrate, α-ketoglutarate, and succinate in the presence of absence of 2,4-dinitrophenol was also investigated. The increase in water content in the brain was 5.30% in the injured hemisphere. Activity of dehydrogenases (I and IV) in the edematous cerebral tissue did not significantly change, though a tendency or activation of II and V within area surrounding the edema focus was observed. III activity in the edematous tissue was increased by .apprx.80%. Phosphatase activity in the edematous cerebral tissue showed a dissimilar evolution; acid phosphatase became more active with an increase of .apprx.35% and alkaline phosphatase was not affected. Compared to the normal tissue, the edematous cerebral tissue oxidized isocitrate in vitro at a slightly higher rate and α-ketoglutarate and succinate at a slightly lower rate. However, in vitro oxidative activity in the edematous cerebral tissue was lesser influenced by 2,4-dinitrophenol than the in vitro oxidative activity of normal cerebral tissue. These observations suggested that the effect of cerebral edema was related to decreased high-energy phosphate metabolism with subsequent alteration of oxidative phosphorylation of some substrates. The changes of enzyme activity produced by an O shortage in the brain were discussed. 25 references.

L22 ANSWER 15 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN

AN 1967:114073 CAPLUS
DN 66:114073
OREF 66:21175a,21178a
TI Enzyme activity in serum and cerebrospinal fluid in normal subjects and subjects with neurologic and psychiatric diseases
AU Amabile, Giuseppe; Pizzo, Paolo A.
CS Univ. Rome, Rome, Italy
SO Rivista di Neurologia (1966), 36(6), 553-61
CODEN: RINEAG; ISSN: 0035-6344
DT Journal
LA Italian
AB Glucose-6-phosphate dehydrogenase (I), leucine aminopeptidase (II), and malate dehydrogenase (III) were determined in the blood serum and cerebrospinal fluid (CSF) of bedridden neurological and mental patients. Blood and CSF were drawn simultaneously and centrifuged 10 min. at 2500 rpm. and 4°. Subjects in good health were used for controls. No correlation between serum and CSF content of the enzymes was found. I, II, and III were found in both fluids. I was highest in CSF in encephalomyelitis and neurosyphilis. II was highest in CSF in sclerosis, encephalomyelitis, and adenoma of the hypophysis. III was highest in CSF in encephalomyelitis. All 3 enzymes were high in chronic alcoholism. Serum I was also elevated in epilepsy.

L22 ANSWER 16 OF 16 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1966:406406 CAPLUS
DN 65:6406
OREF 65:1204c-d
TI Cerebrospinal fluid enzymes in some neurological disorders
AU Tsvetanova, E.; Doseva, I.
CS Bulgarian Acad. Sci., Sofia
SO Nevrolog. Psikhiatr. Nevrokhirurg. (1966), 5(1), 39-48
DT Journal
LA Bulgarian
AB A review of the changes of enzymic activity of aldolase, lactic dehydrogenase, malic hydrogenase, glucose-6-phosphate isomerase, isocitric dehydrogenase, creatine phosphokinase, α-amylase, phosphatases, transaminases, cholinesterase, RNase, and DNase in cerebrospinal fluid during some neurological disorders. 99 references.

=> dis hist

(FILE 'HOME' ENTERED AT 15:23:35 ON 02 NOV 2007)

FILE 'APOLLIT, MEDLINE, BIOSIS, EMBASE, BABS, CAPLUS, CBNB, CIN, COMPENDEX, DISSABS, EMA, IFIPAT, NTIS, PASCAL, PROMT, RAPRA, SCISEARCH, TEXTILETECH, USPATFULL, USPATOLD, USPAT2, WPIFV, WPINDEX, WSCA, WTEXTILES' ENTERED AT 15:26:19 ON 02 NOV 2007

L1 192059 S NEUROLOGICAL AND DISORDER
L2 3665 S L1 AND (MANNOSE OR GULOSE OR GLUCOSE-6-PHOSPHATE)
L3 1090 S L2 AND (CAMP AND MODULATOR)
L4 11 S L3 AND ONCOMODULIN
L5 1062 S L3 AND (STROKE OR ANEURISM OR SPINAL OR PARKINSON OR SCLEROS
L6 832 S L5 AND MACROPHAGE
L7 832 S L6 AND FACTOR
L8 661 S L7 AND TGF
L9 620 S L8 AND ALZHEIMER
L10 10 S L9 AND ONCOMODULIN
L11 619 S L9 AND NEURON?
L12 408 S L8 AND GLAUCOMA
L13 368 S L12 AND INTRAOCULAR

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L14          367 S L13 AND INJECT?
L15         6805 S L1 AND RETINA?
L16          358 S L14 AND RETINA?
L17            5 S L10 AND MACULAR

     FILE 'CAPLUS' ENTERED AT 15:43:13 ON 02 NOV 2007
L18           69 S BENOWITZ LARRY I?/AU
L19            5 S L18 AND NEUROLOGICAL
L20           24 S L1 AND MANNOSE
L21            2 S L1 AND GULOSE
L22           16 S L1 AND GLUCOSE-6-PHOSPHATE
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EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
S1	2845	514/23	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2007/10/31 16:00
S2	1697	S1 and (mannose or gulose or glucose)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2007/10/31 16:01
S3	1700	S1 and (mannose or gulose or glucose$)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2007/10/31 16:01
S4	175	S3 and neurolog$	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2007/10/31 16:21
S5	10	S4 and (macrophare or ionophore or phosphodiesterase or adrenoreceptor)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2007/10/31 16:23
S6	47	S4 and (macrophage or ionophore or phosphodiesterase or adrenoreceptor)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2007/10/31 16:04

EAST Search History

S7	364	S3 and (stroke or aneurism or spinal or parkinson's or sclerosis or alzheimer or atropy or picks or dementia)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2007/10/31 16:21
S8	355	S7 and treat$	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2007/10/31 16:22
S9	24	S1 and (glucose ADJ phosphate)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2007/10/31 16:20
S10	1	S9 and neurolog$	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2007/10/31 16:17
S11	35511	(mannose or gulose) or (glucose ADJ phosphate)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2007/10/31 16:22
S12	2681	S11 and neurolog$	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2007/10/31 16:22

EAST Search History

S13	7625	S11 and (stroke or aneurism or spinal or parkinson's or sclerosis or alzheimer or atropy or picks or dementia)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2007/10/31 16:21
S14	34686	(mannose or gulose)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2007/10/31 16:22
S15	2617	S14 and neurolog$	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2007/10/31 16:22
S16	2602	S15 and treat$	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2007/10/31 16:22
S17	541	S16 and (macrophare or ionophore or phosphodiesterase or adrenoreceptor)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2007/10/31 16:23
S18	1453	S16 and (macrophage or ionophore or phosphodiesterase or adrenoreceptor)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2007/10/31 16:24

EAST Search History

S19	6263	S14 and (macrophage or ionophore or phosphodiesterase or adrenoreceptor)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2007/10/31 16:24
S20	484	S19 and (neurological ADJ disorder)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2007/10/31 16:25
S21	484	S20 and treat$	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	ON	2007/10/31 16:25